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OF

DISEASES OF THE NERVOUS SYSTEM

BY THE SAME AUTHOR

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A MANUAL

OF

DISEASES OF THE NERVOUS SYSTEM

BY

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Third Edition

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VOLUME I

DISEASES OF THE NERVES AND SPINAL CORD

WITH ONE HUNDRED AND NINETY-TWO ILLUSTRATIONS

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PREFACE TO THE FIRST EDITION.

THIS volume contains the first part of an attempt to give an account of diseases of the nervous system, sufficiently concise to be within the compass of the time-pressed student or busy practitioner, and yet adequate in its outline of a subject which has become wide and deep beyond any other part of medicine. Success in both aims can scarcely be more than approximate.

Most of the illustrations are printed from blocks prepared from original drawings by phototype processes, and I am indebted to Messrs. A. and W. Dawson, of the Typographic Etching Company (by whom most of the work has been done) for the care they have taken in the reproduction.

The casual reader may perhaps miss subsidiary letters in the illustrations of the lesions of the spinal cord. The omission of these is intentional. A knowledge of the structural topography of the cord is the first requisite in the study of its morbid anatomy, and when this knowledge is gained, the figures will be understood without difficulty. Familiarity with unlettered illustrations facilitates the comprehension of sections of the spinal cord.

QUEEN ANNE STREET, LONDON
August, 1886.

43456

PREFACE TO THE THIRD EDITION.

In the present edition every chapter has been subjected to careful revision, and numerous additions have been made embodying the results of personal experience and the more important facts ascertained by others.

We are indebted to Dr. Bertram Abrahams for assistance in the revision of the section on the Anatomy and Functions of the Spinal Cord, and to Dr. F. E. Batten for the appendix on the Muscle-spindles.

W. R. GOWERS.
JAMES TAYLOR.

January, 1899.

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DISEASES OF THE NERVOUS SYSTEM.

INTRODUCTION.

A CLASSIFICATION of Diseases of the Nervous System, at once scientific, exact, and convenient for systematic description, is not at present possible. If we attempt to classify the diseases according to either seat or nature, we are met at once by difficulties which prevent any complete arrangement. These difficulties arise from the fact that many diseases affect more than one part of the nervous system, that the precise seat of other maladies is unknown, and that we are still ignorant of the nature of a considerable number of diseases. A rough division is often made into two broad classes of "organic" and "functional" disease. The first class, that of "organic diseases," comprehends those in which there is always a visible lesion, manifesting sometimes the morbid process which constitutes the disease, sometimes only the ultimate result of that process. The second class, that of "functional diseases," is less definite, comprehending (1) those diseases that consist only in a disturbance of function, and are therefore properly so designated; and (2) many diseases which have this in common with true functional disease, that they are transient and not permanent, and that they are not known to depend on organic changes. But there is also a large class of diseases in which no constant lesion has been discovered, but which are not transient, and cannot well be placed in either of these classes. This simple classification is therefore inadequate. It may be well to consider in what respect it needs alteration to meet the requirements of our present knowledge.

The term "organic disease," as commonly used, means visible disease. But visibility is relative; it depends on the means of vision. The application of the microscope to pathology immediately increased the range of organic disease, and a similar increase has resulted, in our own day, from the use of staining agents, which render visible many changes that could not before be seen. Still it remains true that the degree of alteration determines visibility. Molecular changes in nutrition, considered as such, must be colossal to be detected. Such alterations, not sufficient to be seen, but still considerable, probably constitute the morbid process in many diseases that are commonly classed as "functional." Hence we may distinguish a class of "nutritional diseases" as to a large extent distinct from those that consist in a mere derangement of function. While distinguishing these two classes, we cannot entirely separate them; nutrition and function are mutually dependent, and prolonged functional disturbance must determine nutritional change.

Visible disease varies much in nature and degree. Some forms can always be seen with the unassisted eye; they change the shape or colour or consistence of the part. Other forms can be detected only with the aid of the microscope; while many may be seen with the unassisted eye when their degree is considerable, and only by means of the microscope in their early stage or when in slight degree. It is convenient to term such alterations, the visibility of which is altogether a matter of degree, "structural diseases." Thus we may distinguish four pathological classes of disease of the nervous system:

1. *Organic disease* (or *"coarse" organic disease*); such as tumour, hæmorrhage, softening.
2. *Structural disease;* such as most forms of sclerosis.
3. *Nutritional disease;* such as general paralysis of the insane, paralysis agitans.
4. *Functional disease;* such as reflex convulsions, and many forms of hysteria.

In the first class the morbid process always begins outside the nerve-elements themselves. In the second class it may begin in them or outside them. In the third and fourth classes these elements are probably always primarily affected.

Such a classification helps us to obtain clearer views of the primary relations of disease, but is not convenient for systematic description. For this a hybrid system is necessary, in which organic and structural diseases are classified according to their seat,—in the nerves, spinal cord, or brain. With each group may be placed some nutritional or functional maladies of local incidence. The bulk of these diseases are, however, best considered separately, after the organic diseases have been described, since many of them are of wide distribution or uncertain seat.

Classification has two purposes to serve. It serves to bring out the differences in the nature of disease, and thus to define our conceptions and make clear our distinctions. But this purpose is less obtrusive than the second,—to furnish us, as it were, with shelves on which to place our conceptions, and labels with which to ticket them, so that we may find them when we need them and study them in a useful order with the least loss of time.

Even if our knowledge were perfect, it is not likely that any scientific classification founded on natural distinctions would serve the practical needs of an arrangement for *use*. Our present knowledge is unquestionably so very far from perfect as to be inadequate even for a complete natural classification; while an attempt to construct one of this character that shall be also of practical convenience is a mere waste of time. For use, we must divide where we ought only to distinguish; and we have to place near together, for convenience' sake, maladies that should be not only far from each other, but sometimes far from all others.

Yet it is useful to attempt, if not to classify, at least to distinguish the chief pathological groups of the morbid processes that are commonly regarded as diseases of the "nervous system," and are so designated. Unserviceable as a practical division, the attempt affords a general view of the nature of the maladies, including the various influences to which they are due, and therefore by which their nature is determined. The view that is obtained is not less useful because the groups that are, in the mass, well defined, are perceived to have connecting links when their borders are scrutinised, or are even found here and there to blend in places where a demarcation can only be artificial. We perceive thus that elements are common to more than one, and such elements may be thus more clearly discerned, and their importance is more clearly revealed.

The first great distinction, which is indeed a cleavage running through all the varieties distinguished for practical convenience, is that of primary seat. Some diseases begin in the nerve-elements, others outside them, in other structures. These structures have widely different pathological relations. The bones which protect, the fibrous tissues that invest and support, the vessels which convey the blood to or from the nerve-structures, bring the diseases of these structures into connection with most chronic and acute constitutional diseases; the blood itself, altered by numerous and widely different affections, frequently produces disturbance of function or of structure, and multiplies the relations of disease of the nervous system to an extent that is to us indefinite, and potentially is all but infinite. Not only does the blood cause disease by the direct influence of its changed constitution or the morbid agents it bears, but the physical conditions of the circulation, by excess or deficiency of force, and the passage in it of bodies that can obstruct the arteries, involve a direct relation to morbid states of the heart, which is frequently obtrusive.

The affections which constitute connecting links between the two general classes of disease, those in which the nerve-structures suffer primarily and suffer secondarily, deserve a general notice. Some of them present to view certain pathological features which are of great importance, and which we have only lately become able to perceive distinctly if not yet clearly.

The various morbid growths are also, for the most part, diseases only in, not of, the nervous system, although one variety of neoplasm affords another illustration of the connection between the two classes; gliomata connect extrinsic growths with diseases that are primarily of the nerve-structure. The supporting neuroglia, different from the nerve-structures as it seems, and indeed is, consists of a residue of the embryonal tissue from which the nervous elements were developed. In its nutritional tendencies it presents a relation to the nerve-structures which, though definite, is opposite; it overgrows when the latter decay, and thus gives rise to the condition of secondary "sclerosis." Its growth seems normally to be restrained by the vitality of the higher tissue, and becomes exuberant as soon as that of the latter is lessened. It may persist in tracts, large or small, when there is a congenital arrest of development, such as gives rise to the cavities called "syringomyelia;" this persistence is especially important because the tissue may cease to be quiet, may grow and develop into the definite and dangerous condition known as "central gliomatosis." But the neuroglia, as the "connective tissue" of the central structures, shares the lower morbid tendency of the interstitial tissue of all organs. It is by an overgrowth of this, in random foci, presenting at first some of the features of inflammation, that "insular sclerosis" develops, while the neuroglia also takes a chief part in all forms of interstitial inflammation in the brain, cord, and nerves. Its secondary overgrowth in degeneration of the nerve-structures varies in amount and in energy; even when secondary, it seems occasionally to assume an independence which makes it excessive, and an energy which involves some of the features of inflammation.

We have to distinguish from the primary interstitial changes, those acute processes in which the nerve-structures suffer primarily, but with rapidity. When they undergo slow decay we call it "degeneration," but an acute destructive change may take place so rapidly as to resemble "parenchymatous inflammation"—*i. e.* inflammation in which the primary element is in the proper functional structures of an organ. Such changes belong to the group of diseases that are truly of the nervous system, but are not always readily distinguished from the interstitial inflammations, since the interstitial tissue and vessels may participate in the more intense processes. Their course presents all gradations, from the most acute to chronic forms, but even in the former it is often remarkable how small is the share the connective-tissue elements take. These processes have become, of late, very important, on account of the extent and degree to which they can be traced to toxic influences. They constitute, in slow form, the systemic degenerations of the central nervous system, and also, in both slow and rapid forms, the varieties of toxic peripheral "neuritis." The former, as well as the latter, have been extensively traced to the influence of blood-states. But as chronic "degeneration," they are also often due to defective vitality, or imperfect vital endurance, senile or premature, occurring in late life before other structures decay, or as isolated failure soon after development is complete. The latter class is represented by the diseases of which "hereditary ataxy" is a type.

The toxic influences which are thus effective seem, so far as we can trace their nature, to be chemical. They may be metallic poisons—or the simpler chemical substances of organic origin, as alcohol—or complex organic compounds, either received from without or, more often, formed within the body. They are often formed within the body by the organisms that give rise to "acute specific diseases," as has been clearly proved in the case of diphtheria; to the same mechanism many nervous sequelæ of acute diseases are apparently due. These poisons, especially those of organic origin, present the remarkable feature of a special tendency to influence certain nerve-structures, that we are only able to distinguish from others by their difference of function; underlying this must be some minute difference of constitution which renders them more susceptible to the particular chemical compounds. These structures suffer most where their vitality is least,—for instance, the ends of the nerve-fibres,—the parts farthest from the nerve-cell of which they are the processes. Thus many toxic agents act on the extremities of the long sensory or motor nerves in the limbs, causing "degenerative peripheral neuritis," while a similar degeneration of the fibres of the pyramidal tracts of the spinal cord (the processes of the motor cells of the cortex) constitutes "lateral sclerosis." The motor fibres of the limbs are processes of the cells of the anterior grey matter of the cord; the sensory fibres proceed from the cells of the ganglia in the posterior nerve-roots. Of the latter, those which seem most prone to suffer are the afferent fibres from the muscles. In locomotor ataxy these nerves suffer first, apparently from a toxic agent which is a result of syphilis, and acts also on the other processes of these ganglion-cells—those which become the fibres of the posterior median columns. These ascend the cord, but depend for their vitality on the cells of the posterior ganglia. Thus the degeneration in the spinal cord, the "posterior sclerosis," and the changes in the peripheral nerves, are but parts of the toxic effect on one set of "neurons." The afferent nerves from both skin and muscle suffer, moreover, from many other toxic causes, and, alike in alcoholic neuritis and in diphtheritic palsy, we may have a close resemblance to locomotor ataxy. A corresponding lesion underlies the correspondence of symptoms. The special susceptibility of the muscle nerves is the cause of the frequency, and early date, of the loss of the knee-jerk, and of the significance of the symptom.

Other toxic agents influence, not the peripheral ends of the chief processes of the cells, but the structures in which nerve-energy is produced in the central mechanism. Such, for instance, is strychnine, and the strychnine-like products of the organism of tetanus; while one element in the production of the curious disturbance of chorea seems to be the action on the elements of the motor cortex of some poison allied to, but not identical with, that which causes acute rheumatism, and is probably a complex organic chemical compound.

Relative deficiency in the power of resisting toxic agents must be ascribed to imperfect vital energy, a lower degree of the energy of life that maintains nutrition. Such imperfection is manifested by a class of cases in which failure of nutrition occurs spontaneously in certain structures, either in late life, as in many cases of senile muscular atrophy, or labio-glossal paralysis, or senile brain failure, or paralysis agitans,—or else soon after complete development is achieved, as in the so-called "hereditary ataxy" and its congeners. Moreover it is possible that a slight difference in the power of vital endurance in some structures, causes a greater readiness to suffer degenerative changes in middle life under acquired influences.

The toxic agents which can thus act on the nerve-structures and impair their nutrition and function are numerous and varied. They may be derived directly from without, may be produced within the body by the organisms of disease, and may be produced by disordered chemical processes apart from external influence, or by an external influence that becomes effective through a peculiar predisposition. They present relations to those rheumatic poisons which act on the fibrous tissues and on other structures. Exposure to cold, apparently the same, may cause in different persons—a general catarrh, local inflammation (as tonsillitis), acute rheumatism (with or without endocarditis), spinal myelitis, or peripheral neuritis, apparently according to the nature of the toxic agent generated, determined by the constitution of the individual. A slight difference in the initial effect may determine a divergence in the chemical processes under the influence of life, and the ultimate result is the same in no two individuals, and may be very different.

GENERAL SYMPTOMATOLOGY.

SYMPTOMS AND THEIR INVESTIGATION.

THE nervous system is almost entirely inaccessible to direct observation. The exceptions to this are trifling: the termination of one nerve, the optic, can be seen; some of the nerve-trunks in the limbs can be felt, either in the normal state or when enlarged by disease.* As a rule, the state of the nervous system can be ascertained only by the manner in which its work is done, and morbid states reveal their presence by the derangement of function which they cause.

The functions of the nervous system are often divided into mental, motor, and sensory; but they are far more extensive than those words denote. The functions of all the organs of the body, all states of the blood-vessels and hence of the circulation within them, and even all processes of nutrition, are under the control of the nervous system. The disturbance of nerve-function produced by disease is conterminous in extent with the human organism. Our knowledge of these symptoms is constantly increasing in range and in degree. They differ, however, very much in relative importance. Some occur more frequently than others, and are more direct in significance. Many have a special relation to certain parts of the nervous system, while others occur in diseases of all parts. It may be well, at the outset, to describe the characters of some of the most frequent symptoms; while the more special symptoms can be conveniently considered when we deal with diseases of that part.

MOTOR SYMPTOMS.†—Disturbance of motion is one of the most common effects of disease. Movement is produced by muscles, but the muscles are excited to contraction by the nervous system. Most alterations of motility, both defect and excess, are due to its derangement. The conditions are very few in which motor changes originate in the muscles themselves. Defect of movement is of muscular origin only in some cases of primary alteration in the nutrition of the muscles. When there is a general impairment of nutrition, the muscles participate in this; they are ill-nourished, and therefore feeble. Such a condition is met with in chronic wasting diseases, in anæmia, and as an acute affection in all acute febrile diseases. This is the chief cause of the muscular weakness that so often results from fevers; it may even proceed to the degree of causing visible changes in the aspect of some fibres.

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* To the new X rays, unfortunately, the nerve-centres are opaque and inaccessible, and the nerves differ too little in transparency from the other tissues to permit them to be seen, while their course is seldom such as to bring them into conspicuous distinction from other structures.

† Some modification of the word kinesis (akinesis, &c.) is sometimes used to designate motor symptoms. These words seem to me to add nothing to our knowledge or to its clearness—certainly nothing to compensate for their cumbersome character; and the definite addition all new words make to the information that has to be acquired is not always sufficiently weighed against their supposed advantage.

In a few chronic diseases the muscles undergo a primary alteration. One is the disease known as "pseudo-hypertrophic paralysis," a special form of a wider malady, in which there is atrophy of the muscles, due to their own defective power of vital maintenance. Rheumatic and other inflammations, commonly interstitial, may greatly impair their power, first by the pain contraction causes, and then by damage to the nerves in this tissue, a state to be distinguished from the primary affection of these endings themselves. In almost all other conditions, loss of power in the muscles is dependent on the state of the nervous system, either directly or through defective nutrition of the muscles, or both. The nutrition of the muscular fibres depends on the motor nerves, although we are still ignorant of the precise nature of that dependence. The subject will be referred to again in the special account of the changes that result from injuries to the nerves.

So also with increased and involuntary muscular action. We know little of what has been termed "idio-muscular contraction,"—that is, contraction originating in the muscle itself. Whenever the whole of a muscle contracts, the contraction is due to an influence acting on all the fibres, and this is generally from the nerve-centres. Even muscular "tone" is dependent on the connection of the muscle with the nerve-centre, since it ceases when the connection is destroyed. It is possible that the extreme degree of the contraction in cramp is the effect of a tendency in the muscle itself. When the shortening exceeds a certain degree it becomes fixed in a peculiar manner, which seems as if it were due to a tendency in the fibres to pass beyond the power of relaxation. Contraction of the whole of a muscle may also result from mechanical influences acting on every part equally, as in traction. But these exceptions occur under special conditions.

The partial contractions that are termed "fibrillary" are probably local, since they are so readily produced by mechanical stimulation, such as a tap on the muscle. Yet fibrillary contractions, although sometimes local, may be often of central origin. This is also true of the flickering contractions that are so common in persons whose nervous system is enfeebled, the "live flesh" of popular language. They may occur in any muscle, from the orbicularis palpebrarum (in which they are most common) to the gluteus maximus. After damage to the nerve-fibres to the muscle they occur in special degree; they are often frequent for years in the calf muscles after sciatic neuritis.

Paralysis.—Defect of movement may present every degree from slight weakness to absolute loss. There may be inability even to make the muscle contract, or the voluntary contraction may occur, but be too feeble to move the parts to which the muscle is attached. The latter difference is to some extent relative, since it varies with the readiness with which the part can be moved—*i. e.* with the resistance to be overcome. A contraction that will flex a finger may be insufficient to flex the wrist. The difference may vary in the same part according to position. A degree of power in the flexors of the hip, that will move the leg when the patient is lying on his side, may be insufficient to do so when the patient is lying on the back. This difference is even more conspicuous in some other muscles; paralysis of the extensors of the elbow may be discovered only on an attempt to raise the hat: in many positions extension is due to the weight of the arm, much more than to the muscles, so that considerable weakness may be unobserved.

The term "paralysis" has long been applied to all degrees of defect of power, a distinction being made between complete and incomplete paralysis. Partial loss of power is also sometimes termed "paresis." The advantages of "paresis" over the word "weakness" are chiefly psychical, and depend on the popular horror of "paralysis," which has become specially associated with complete loss of power, and the idea of incurability seems inseparably attached to the term.

Our means of estimating defects of muscular contraction are very imperfect, in consequence of the peculiar and various forms of movement which are produced. An instrument to measure the force exerted is called a "dynamometer," but any simple form of instrument can have only limited application. Several have been devised, but that in general use consists of an oval steel ring, which can be narrowed either by compression applied to its smaller diameter or by traction applied to its longer diameter. The change in the shape of the dynamometer, and therefore the force exerted, is shown by the movement of an index on a scale that has a double graduation, a closer for compression, which is easy, and a wider for traction lengthways, which is difficult. This form of dynamometer is known as "Charrière's," from the name of its first maker. Another less convenient form was devised by Duchenne. These instruments are chiefly used for the measurement of the grasp. The estimation of the strength of other movements is practicable, but not easy. The observer opposes the movement by means of the instrument, and notes the resistance that can be overcome. Thus pressure applied through it just above the patella, to force down the raised thigh in the sitting posture, shows the power of the flexors of the hip. The wide variations in strength among different persons renders absolute estimation possible only when the defect is considerable. Slight changes are recognised by comparing the power on the two sides. It should be remembered that the limbs on the right side are normally stronger than those on the left, the relation being about as five to four. Care must be taken to secure uniformity of the conditions under which the observations are made. A careful search would doubtless reveal average proportions between other movements that can be measured. Thus the strength of flexion of the hip, estimated in the way just mentioned, often corresponds to the strength of grasp on the same side.

Inco-ordination of Movement.—By this term is meant a defect in the relative time and degree of the muscular contractions by which a movement is produced. Every movement is due to the contraction of a series of fibres, which may or may not correspond to the series massed together in a "muscle." Movements, not muscles, are represented in the structure of the brain, and the fibres which, together, cause a simple movement may be in more than one muscle, or may be only part of a muscle. The *synergic* action of the several fibres, in a definite degree and at a certain time, is essential for the movement. So also is the simultaneous contraction of the opponents of these acting muscles, which are thus steadied; this *antergic* * contraction is essential for the exertion of force, and must be often great in degree when strength rather than amount of movement is required. It must also be lessened automatically to permit movement. (The process will be considered in connection with the functions of the spinal cord.) Defect in the opponents does not interfere with *movement*, but it prevents the exertion of force, by permitting the movements to be too great.

It is not customary to apply the term "inco-ordination" to any derangement of movement except that which is irregular in time. There may be also irregularity in degree, but it is when the contractions have not the normal duration, or are not synchronous, or not in due sequence, that the conspicuous irregularity occurs to which the term is applied. This is also often called "ataxy."

Its manifestations differ in character according to the character of the movement. The variations are so great that it is not practicable to describe them, nor is it necessary, since all have the general nature just stated. It is also not easy to estimate the inco-ordination so as to permit definite record. When the movement of the arm is affected, the degree may be estimated by making the patient try suddenly to touch, with the point of a pencil, a spot on a sheet of paper placed before him, his eyes being closed at the moment of the attempt. After a certain number of attempts the average distance from the spot of the dots he has made is ascertained, and this, compared with the result given by a normal individual, affords a numerical indication of the degree of inco-ordination.* The method is effective, although the result is perhaps scarcely worth the trouble.

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* I use this term because we have none to express antagonistic co-operation. This is synchronous with that of the acting muscle, but not synergic.

Whenever there is inco-ordination, spontaneous movements occur if the patient tries to hold the limb in a certain posture; the variation in muscular contraction prevents fixation as well as disorder in movement; co-ordination is as essential for steadiness of uniform contraction as for the steadiness of the varying contractions of movement. These involuntary movements constitute a delicate test for the presence of ataxy.

All spontaneous muscular contractions derange those that are due to the will, and movement is disordered by any "tremor" which co-exists. The slight rapid contractions that cause "tremor" may, indeed, occur only as voluntary movement, and there is a gradation of disorder from simple inco-ordination to that which may be called "tremulous." So also more extensive spasm disturbs movement with which it coincides, and energetic contractions of involuntary nature may be excited by a volitional impulse. Hence, just as simple ataxy may pass into tremor, so, on the other hand, there is a gradation by "jerky inco-ordination" to wide sudden movements, the violence of which obscures the element of mere disorder.

It is necessary to distinguish *co-ordination* and the *direction* of movement to a definite end. The motor process for the latter depends on sensory guidance, chiefly on the senses of touch and sight. A sempstress with loss of sensation in the fingers cannot sew unless she constantly watches them. A compositor, whose sense of touch is dulled upon the tips of thumb and index finger, cannot pick up the type, or if he does pick it up may drop it again, unless he constantly watches the movement. There is also a guidance from the afferent nerves of the muscles, but this is related to co-ordination proper rather than to the direction of movement, and it will be more conveniently considered in connection with the functions of the spinal cord. Indeed, the mechanism of co-ordination, and its special factors, can only be discussed in that connection.

Spasm.—Involuntary muscular contraction, when considerable in degree, termed spasm, is always due to the influence of the nervous system. It is a frequent symptom of disease. Three chief varieties may be distinguished, but intermediate and combined forms are often met with. (1) We may have, first, a single isolated very brief contraction of one or more muscles. (2) Separate contractions may occur in rapid succession, a second commencing as soon as there is relaxation of the muscle from the first contraction. This is termed "clonus," or "clonic spasm." The relaxation of one is often incomplete when the next contraction begins. (3) There may be a persistent contraction in which no relaxation can be perceived. This is termed "tonic spasm."

(1) It will be remembered that muscles, as long as they are connected

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* Blix, 'Neur. Centralb.,' 1884, p. 83.

with the nerve-centres, are never in a state of perfect relaxation. There is always a certain degree of persistent slight contraction of the fibres, which keeps them adapted to the distance between their attachments, so that the substance of the muscle presents always a similar degree of firmness. This physiological "tone" is due to a continuous action of the centre on the muscles, through the motor nerves; but this action is apparently determined, in degree at least, by the sensory impressions from the parts, and especially from the muscles.[*] Tonic spasm may be regarded as an augmented degree of this state, due to various causes. Although it necessarily depends directly on the action of the centres in the spinal cord, this may be due to the augmenting influence of the cerebral centres, or to diminution of the latter, permitting insubordinate activity, and is often clearly the result of a reflex process. It must be distinguished from structural shortening of the muscle. The distinction is easy, because tonic spasm can always be overcome by gentle, steady traction, while structural shortening cannot be thus removed. The latter may result from long-continued tonic contraction; nutritional changes alter the structure of the muscle and fix the elements, especially the interstitial tissue, in the state of contraction. It occurs whenever muscles for a long time are unextended by their antagonists, and so results from the long-continued maintenance of a certain posture, and also in extreme degree from paralysis of opponents. Its influence on the joints and the parts moved causes the various deformities that result from local palsy. The tonic spasmodic shortening which can be overcome is often spoken of as "spastic." It is sometimes also distinguished as "contracture," the term "contraction" (which really includes all forms of spasm) being then applied in a narrow sense to the fixed shortening.

(2) Single muscular contractions vary much in extent and duration; when partial they sometimes, as already stated, seem to be of local origin. When more extensive they depend on the nerve-centres.

(3) Clonic spasm consists of a series of brief contractions, each of which corresponds to that which is caused by a single induction-shock applied to the nerve. This has a definite duration of one tenth to one twelfth of a second, but it is often prolonged by the force which the contracting muscle has to overcome. In the serial contractions to which the term "clonus" is applied there is also an interval between the end of one and the commencement of another, occupied by the process of stimulation. The "foot-clonus" to be presently described, which consists of a series of such contractions, varies in frequency from five to eight or ten per second. The contractions in "tremor," as seen for instance in "paralysis agitans," have a somewhat greater average frequency.

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[*] Jendrassik ('Neur. Centralbl.,' 1896) has endeavoured to estimate the degree of tone by the resistance of the muscles to passive extension, and finds it greater in spastic paraplegia, and less in tabes than in normal states. The latter, however, presented such wide variations as to preclude absolute inferences. The amount of the extending force applied seems to need more careful adjustment.

Tetanic spasm is produced by the blending of clonic contractions which may be so fused that the contraction is uniform, and is then identical with tonic spasm; or there may be commencing relaxation of one before the next contraction supervenes. This is chiefly seen as such spasm develops or lessens. The passage of tonic into clonic spasm is well seen in the severe epileptic convulsion, in which the fixed spasm becomes first vibratory, then intermittent, and at last remittent.

Sensory Symptoms.—Altered sensation is another very common symptom. It has often to be searched for, because a significant defect may be unknown to the patient. Each form of sensation, touch, temperature, and pain, must be separately tested, since one may be affected and not another. The affection of one form only is sometimes of important diagnostic significance.

Sensibility to Touch.—The structures that subserve tactile sensibility are stimulated chiefly by motion—by change in contact. Prolonged contact, uniform in its degree and place, is perceived but little, and only by close attention. It is the change that excites a nerve impulse; the commencement of contact is more effective than its cessation, but in each case rapidity of change chiefly determines the result. a firm pressure may be scarcely noticed if produced very gradually, while a sudden touch is observed, however light. In testing tactile sensibility care must be taken that the instrument employed does not give an impression of heat or cold, lest the patient perceive by the sense of temperature that which he cannot perceive by the sense of touch. The observer's finger may be used, if it is not cold, or the skin may be touched with a feather, a piece of string, of india rubber, &c. The eyes of the patient should be closed during the examination, and, since he is apt to fancy that he feels a light touch when he does not, he should occasionally be asked if he can feel, when no contact is made. But it is easy to make the test too fine. Where the skin is thick, or sensibility is normally low, a light touch with a piece of string or a feather may normally be unperceived. Loss of sensibility to touch is termed "anæsthesia," but this word is often applied loosely to all forms of impaired sensibility.

In examining the tactile sensibility it is important to ascertain not only whether the patient can feel, but whether he is able to recognise the place touched,—whether he can correctly "localise" the sensation. For this he must be asked not only whether he feels the touch, but also to say or point out where he feels it. The part touched should be frequently varied. Another test for tactile sensibility depends on the fact, ascertained by E. H. Weber, that the distance apart at which two points are discriminated is nearly the same in different individuals in the same part of the body, although it varies much in different parts of the body. The normal distance being known, the increased distance at which the points have to be placed for their discrimination is a measure of the degree of defect. The points should not be so sharp as to occasion pain. Common compasses may be employed, but the most convenient "æsthesiometer" is one contrived by Sieveking, in which the points are attached to a graduated bar. The greater the degree of tactile sensibility, the nearer together can the points be, and still be discriminated. The distance at which they are normally distinguished must be known before any inference can be drawn. The most important average distances, ascertained by Weber, are as follows:— The distance is smallest, *i. e.* the sensitiveness is greatest, at the tip of the tongue, where the points are discriminated when only 1·5 mm. apart. Then come, in order of sensitiveness, the finger tips, 2 to 3 mm.; the lips, 4 to 5 mm.; the tip of the nose, 6 mm.; the cheeks and the backs of the fingers, about 12 mm.; the forehead, 22 mm.; the neck, 34 mm.; the forearm, lower leg, and back of foot, 40 mm.; the chest, 45 mm.; the back, 60 mm.; the upper arm and thigh, 75 mm. Slight variations exist in different individuals, and a deviation from the normal that is uniform in degree throughout the body is probably physiological. In using this test it is necessary to touch the skin with the two points at the same moment, and with equal pressure. The examination requires time and patience, and the results are considerably modified by the intelligence of the patient. Moreover the power of discrimination is increased by practice. It is rare to obtain a conclusive result unless there is a degree of defect that causes a slight absolute loss. For these reasons the practical value of the test is less than was at first anticipated, and it is not often employed. It is most useful for the estimation of changes of sensibility in the same person and the same part.

Curious modifications of tactile sensibility are sometimes observed. In one of these, a single touch is felt as if it were two or even three, a condition that has been termed "polyæsthesia" (Fischer). In another, an impression on one part is referred to some other part, usually in the same limb, rarely in another limb.* In another variety, an impression on one part has been referred to the corresponding place on the opposite side of the body; this has been termed "allocheiria" (Obersteiner).

Perception of the degree of pressure on the skin is probably subserved by the nerves for tactile sensibility. It may be estimated by simple pressure applied through any instrument, or by small weights, or by an instrument contrived for measuring the tension of the pulse in which, by means of a spring, the part tested must be supported, so that the muscles are not brought into action. The point to be ascertained is the minimum variation that can be recognised. In health this is about one twentieth of the total pressure, whatever the latter may be (Weber). The sensation in the skin caused by electricity, especially by faradism, does not always correspond to that due to other stimuli. It has been thought to depend on a special form of sensibility, but the evidence of this is insufficient. Its features and their meaning have yet to be ascertained.

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* In most persons there are spots in the skin whence a painful sensation seems to dart to a distant place; *e.g.* a prick on the thigh in a limited area may be felt also as a sharp pain near the scapula. This shows how structural connections must exist favouring the "reference" of sensation to a distance from the nerve stimulated. Another instance of the same perverted reference is presented by the frequent neuralgic pain in one jaw due to a carious tooth in the other jaw, which seems painless.

Sensibility to pain is subserved by what are called nerves of "common sensibility." It may be tested by a prick or a pinch. For a prick, too fine a point must not be used, not only because a sharp point may penetrate the skin, but because, in the less sensitive parts of the skin, where the nerve-endings are not close together, a fine point may be unfelt at one spot, although felt readily at a place close by. Nothing answers better than the point of a quill pen. The faradic current may be employed to test sensibility to pain, wire terminals being most suitable for the purpose. Its advantage is the delicacy with which it can be graduated; but it does not furnish any absolute standard, and needs more study before it can be usefully employed.

Sensibility to pain may be changed with or without sensibility to touch. Its loss is termed "analgesia," but is often included in the general term "anæsthesia." A painful sensation may be felt more intensely than normal; this is usually called "hyperæsthesia"—a general term for increased sensitiveness; sometimes, with more precision, "hyperalgesia." Occasionally a touch on the skin gives rise to pain, but it is probable that this is due to the stimulation of the over-sensitive nerves of common sensibility, and is not an intensification of a tactile sensation. The fact that touch may be perfect, although no pain can be caused, makes it difficult to conceive that the nerves of touch can subserve pain. Both tactile and painful impressions may produce sensations that are abnormal in character, described as "thrilling," "tingling," &c. This perverted sensation has been termed "paræsthesia," or "dysæsthesia," words that have also been applied to purely subjective sensations.

When a prick is perceived as touch, pain being lost, care is needed that the patient shall clearly know what is desired. Since there is often delay, when there is not loss, he should be told to say "touch" or "prick" the instant either sensation is perceived. When sensibility to pain is diminished but not lost, the test must be kept uniform. Another source of error is then occasioned by the readiness with which the patient may mistake the degree of pain he is required to recognise, and by the fact that his idea often varies in the course of the examination. He may thus deny that he feels a prick as such at a part where presently he says he does, and this quite honestly, the sensation being really the same. It is probable, indeed, that this is the most frequent cause of the conclusion that the state "varied in the course of the examination," or that "the patient's answers were unreliable."

Sensibility to temperature is usually affected with sensibility to pain,

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although the strange fact that they contain striated muscular fibres makes it probable that motor fibrils may enter them. Their relations, degenerative and morbid, have yet to be worked out, but we may assume that it is probably in them that the afferent impulses arise. Such impulses are excited especially by pressure, and by traction on the muscle. Thus a squeeze of the calf causes a muscular sensation of an undeniable quality, and strong passive flexion of the foot on the leg causes a sensation which is distinctly produced *in* the muscle. The nerves are, moreover, strongly stimulated when the fibres are widened by the extreme contraction of "cramp," which causes acute pain. It must be ascribed to the compression of these nerve-endings, since no afferent impressions seem to come from within the fibres. It is thus a proof of their susceptibility to pressure-excitation. After cramp, moreover, the nerves are left in a state of such exalted excitability that even slight extension of the muscle gives rise to pain, although such extension has no sensory effect in the normal state. Every one has experienced the effect. We shall afterwards see the importance of the fact that the nerves are chiefly stimulated by these two mechanical processes.

The muscular pain caused by electrical stimulation, as distinct from the pain felt in the skin, may be due to the stimulation of the afferent nerves in excessive degree, or to the effect on them of the muscular contraction that is produced; the former is the more probable. But these sensations of pain, although due to "muscular sensibility," are not included in the term "muscular sense." They are, however, of great importance on account of the conclusive evidence they afford that afferent impulses come from muscles and as to the way in which they are produced.

The chief knowledge that is ascribed to the "muscular sense" is that of the character of movement and posture due to the action of the muscles, of the degree of movement and energy of contraction, and also of the character of passive movement (by an external force) and of posture when the muscles are at rest. It is evident, however, that here we have two very different conditions, in one of which there is much activity of all the motor elements, nervous and muscular, while in the other these are almost at rest, only such gentle action persisting as maintains the "tone." Except in absolute relaxation, there is probably continuous stimulation of the afferent nerves by this tonic state of the muscular fibres. In active contraction, this stimulation is far greater, and activity of the motor nerve-centres is added. Hence two elements are comprehended in the term, and must be separately considered.

Our knowledge of active states of the muscle is due, at least in some measure, to the effect on consciousness of the activity of the nerve-structures causing the movement. The proof of this consists in three facts. (1) In palsy of an ocular muscle, objects seen are referred to the position (in relation to the body) that they would occupy if the movement corresponded to the innervation; it is the latter, *i. e.* the activity of the centre, to which the perception corresponds. (2) After amputation of a limb, a person who makes an effort to move the lost part seems to feel as if he did move it. (3) In some convulsions beginning locally, slight attacks may be attended with a feeling that the arm is raised above the head, or otherwise moved, when it is hanging by the side. These facts show that the central motor process is an important source of our knowledge. But to its effect must be added that of the impulses from the muscles, about to be described.

Our knowledge of rest-posture and passive movement must be derived from incoming impulses. These are not from the skin; the sense of posture may be lost when cutaneous sensibility is normal, and perfect when this is much impaired. This is observed in unilateral lesions of the spinal cord, and in other diseases. The chief source of these perceptions must therefore be the deeper afferent nerves, those of the muscles and joints, perhaps chiefly of the muscles.

These afferent impulses are no doubt continuously generated, but do not influence consciousness as definite sensations. They certainly influence the activity of the motor centres of the spinal cord, and there is reason to think that they pass, in part, to the cerebellum, and act through it on the cerebral cortex, determining and assisting in regulating its activity. This influence on the motor centres, by the effect it produces, is apparently one chief cause of the perceptions of these passive states. The active state of the motor centres of the cortex, however it is produced, has an effect on consciousness and enters into perceptions though no sensation results. This may be combined either with true sensation from the parts concerned, or with the result of still slighter impulses, to be discerned only by close attention, to the joints, skin, &c., of which we seem, at first, to be altogether unconscious. We are thus led to refer our perceptions of passive posture in part to the same structures as give rise to our perceptions of active posture. Our knowledge is the result of such perception, and not of true sensation proper;—we perceive and know that which we do not feel.

Our recognition of resistance to contraction affords one of the most delicate tests for "muscular sensibility." It is ascertained by testing the ability to detect differences of weight, *i. e.* variations in the resistance to contraction. In this examination the patient's eyes should be closed, and the objects used should be of uniform size. Leather balls like small cricket balls, containing various weights from two drachms to two pounds, are in use at the Queen Square Hospital for this purpose. To eliminate as far as possible the stimulation of the cutaneous nerves, we may (1) reduce it to a minimum area, placing the weights in a bag suspended by a string to the part to be tested, so that only a small area of the skin shall be pressed upon; (2) increase the area (Hitzig), and thus diffuse it in diminished degree; (3) increase the weight, and therefore the pressure in both area and degree, so that the addition to be discerned shall bear only a small proportion to the total stimulation.

Thus we may test muscular sensibility:—(1) by the power of discriminating weight. This is greater than in the case of the nerves of the skin, since a difference of one fortieth of the total weight can be recognised under normal conditions; (2) by the sensitiveness of the muscles to pressure and traction; (3) by their sensitiveness to electrical stimulation. It is difficult to test accurately the electro-sensibility unless the sensitiveness of the skin is lost, or is removed, as by the injection of cocain.

The power of recognising passive posture is also regarded as a test for muscular sensibility, but it may mean much more, afterwards to be considered. If cutaneous sensibility is normal, the parts handled must be grasped firmly, and pressed on both sides, so that the direction of pressure may not suggest the posture. The patient should be asked to indicate the sensation by putting the limb of the other side into the same posture. Several observations should be made to eliminate the chance of accidental error.

REFLEX ACTION.—Many symptoms of disease of the nervous system are due to derangement of the various reflex actions. These are numerous, and we can now only consider their general characters. Each action is effected through afferent sensory nerves, efferent motor nerves, and a system of nerve-cell-processes, and intervening substances,—the "reflex centre." The centre is usually complex and often extensive, and in it are paths of different "resistance," determining the form of reflex action and its extent, according to the source and intensity of the sensory impression. The reflex centre is between the roots of the sensory and motor nerves concerned, and with them it constitutes what may be termed a "reflex arc." The sensory impulse may not only excite a motor process, it may also pass up to the brain, and influence consciousness as a sensation. It is probable that the two effects are subserved by the same nerve-fibres, so far as the cutaneous nerves are concerned, but some fibres from the muscles pass up the cord without connection with the spinal centres. Further, the reflex centre of the spinal cord is to some extent under the control of encephalic centres—a point of very great importance.

Two forms of reflex action must be distinguished. The first is that excited chiefly by stimulation of the nerves of the skin; the second is produced by the stimulation of deeper nerves, chiefly those of the muscles, in the manner just described. This form is caused, among other ways, by tapping a tendon, and therefore was at first, and still is sometimes, called "tendon-reflex action"—an undesirable name.

Cutaneous Reflex Action.—The reflex movements obtained from the skin are excited rather by a gentle stimulation, as a touch, than by a strong, painful impression. They consist of a single movement in most instances, either quick or slow, but often complex, and extensive in proportion to the character of the stimulation effected, the nature of the central mechanism, and the state of the centre. Often muscles at a distance are influenced by a powerful stimulation, either with others or alone. A strong impression may cause a reflex action so wide as to involve most of the muscles of the body. As a rule a painful impression causes quick flexion of a limb, such as to withdraw it from the cause of the pain. Thus a prick near the knee causes a reflex flexion of the hip. In other cases, where withdrawal is impossible, a protective movement results, as in the case of the eyelids.

The slighter degrees of excitation cause a contraction in the muscles close to the part of the skin that is stimulated, the impulse passing, in the spinal cord, only to the related motor centres. This limitation of the process renders them important in diagnosis, especially when lost on one side (see the chapter on the functions of the spinal cord). The cutaneous reflex actions may be excited at almost any part of the skin, but at some parts they are very definite in character, and are distinguished by special names. The most important are the "plantar reflex," from the sole; the "gluteal reflex," a contraction in the gluteus when the skin over the muscle is stimulated; the "cremaster reflex," a retraction of the testicle on stimulation of the skin on the inner part of the thigh; the "abdominal reflex," in the muscles of the abdominal wall when the skin over the side of the abdomen is stroked; the upper part of this reflex is a very definite contraction at the epigastrium, and has been termed the "epigastric reflex." A series of reflex actions may be obtained in the muscles of the back, the highest being in the muscles of the scapula. Others have been distinguished and named, but this process may easily be carried too far. These spinal reflex actions vary in their excitability in different individuals, and are always more readily produced in the young than in the old. They may be increased or abolished by disease.

In the region of the cranial nerves the most important reflexes are those of the eye,—(1) the conjunctival reflex; (2) the contraction of the pupil on exposure of the eye to light; (3) dilation on stimulation of the skin of the neck.

Muscle-reflex Action; "Tendon-reflexes."—The second group of phenomena which depend on reflex action were first systematically studied by Erb and Westphal more than twenty years ago. Attention was called to them in this country by Grainger Stewart and Buzzard in [illegible]. They have since been the subject of a vast amount of study and [illegible]. All occur in muscles that are in a state of slight tension, and are produced by a sudden increase of this tension or by some other sudden mechanical influence, but voluntary contraction superadded to passive tension, diminishes or prevents them. They are distinguished according to their seat, and vary in distinctness, conspicuousness, and the ease with which they are produced according to the mechanical conditions of the muscles and their attachments.

The [illegible] of these is the jerk of the leg which occurs when the patellar tendon is tapped. It has been called the "knee-phenomenon" by Westphal, the "patellar tendon-reflex" by Erb, the "knee-jerk" by myself. To obtain the jerk, the knee must be flexed so that the quadriceps femoris is gently extended. If then the patellar tendon is struck, the quadriceps contracts, and the lower leg is jerked forward if free to move. The blow is given over the space above the tibia where the tendon can yield, so that there is a sudden increase in the tension of the muscle. The most convenient position is with the knee to be tested flexed nearly, but not quite, at a right angle, by being placed over the other knee as the person sits (Fig. 1). But if the leg to be tested is stout, its tension in this position may be too great to permit of any movement. In such case the observer may place his arm beneath the patient's thigh, just above the knee, and rest his hand on the patient's other knee (Fig. 2). Muscular relaxation is sometimes more readily obtained when the legs hang vertically; or the foot may rest on the floor, and the contraction be felt by the hand placed on the muscle; the effect of the recoil and of the muscular contraction must then be carefully distinguished. If the bent fingers of each hand are hooked together, the hands pulled strongly, and the eyes closed, the movement is increased in degree—"reinforcement," it has been termed.[*] It is essential that the flexors also should be free from voluntary contraction. This contraction may be ascertained by feeling the hamstring tendons, and pressure on these by the fingers often helps to secure relaxation. The blow may be given by the side of the hand, a percussion hammer (Fig. 2), or a stethoscope with an india-rubber edge to the ear-piece. If the jerk is doubtful, the skin should be bared.

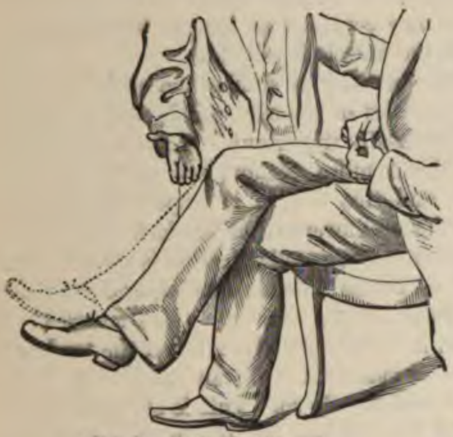

FIG. 1.—THE KNEE-JERK.
The dotted line indicates the movement which follows the blow on the patellar tendon.

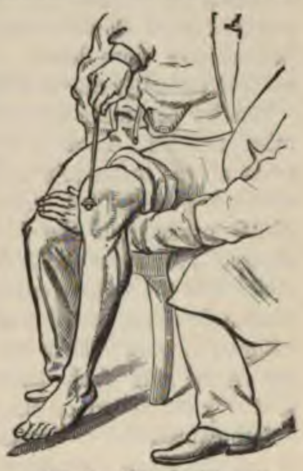

FIG. 2.—THE KNEE-JERK.
Method of obtaining it when it is not readily produced in the ordinary way.

[*] A useful device suggested by Jendrassik.

The same contraction may be obtained by other modes of suddenly increasing the traction on the muscle, especially when this excitability is abnormal in degree. A blow on the tibia may be effective, but a method which has much practical convenience is this. When the patient is lying with the muscles relaxed, the patella is displaced downwards by a finger placed across its upper edge; a tap on the finger in the direction of pressure, so as suddenly to increase the tension, excites the contraction of the muscle whenever there is even slight increased excitability. If the tension is maintained when the excitability is much greater than normal, the single contraction is immediately succeeded by a second, and this by a third, and so on—a series of quick clonic contractions, or "clonus," which will be considered presently in more detail. It may continue as long as tension is kept up, but instantly ceases when the muscle is relaxed.

Similar contractions can be obtained by similar measures in many other muscles. That in the muscles of the calf, which causes a movement at the ankle-joint, is especially important. If the calf muscles, which are connected with the Achilles tendon, are made tense by gently pressing up the foot, and this tendon is tapped, the muscles contract, causing a slight extension movement of the foot; just as the muscles of the thigh contract when the patellar tendon is struck. The tendon is unsupported just as is the patellar tendon, and so yields before the blow increasing suddenly the tension on the muscles. A tap on the side of the tendon is equally effective; but if the tendon is so firmly supported on the other side that it cannot yield, a tap which before caused the contraction will not do so. This is evidence that it is not the mere percussion of the tendon, but the increased tension, which constitutes the stimulation. In cases in which the excitability is excessive—just as sudden tension in the thigh muscles will cause a contraction, followed by others in a continuous series—so, in such cases, if the calf-muscles, which extend the ankle-joint, are suddenly put on the stretch by pressing the hand against the sole of the foot (Fig. 3), the first contraction is followed by another, and they recur as long as the tension is maintained. This series of contractions, the "foot-clonus" or "ankle-clonus" (or "foot-phenomenon"—Westphal), is a very important symptom. The movement is remarkably uniform under the same conditions, varying from six to nine contractions per second. By attaching a writing point to the foot, and making it

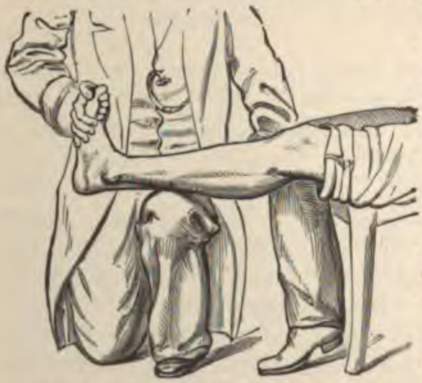
FIG. 3.—METHOD OF ELICITING THE FOOT-CLONUS.

trace a line on a revolving cylinder covered with blackened paper, tracings may be obtained (Fig. 4), which are almost as regular as

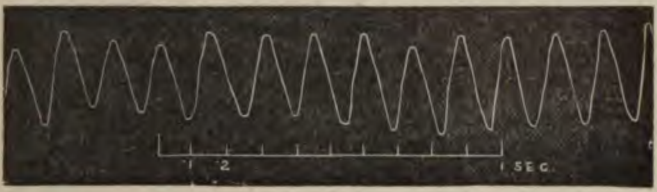

FIG. 4.—TRACING OF THE FOOT-CLONUS IN PARAPLEGIA.
(The tracing reads from right to left.)

those of a tuning-fork.* The clonus in the extensors of the knee has the same time, and the two are of the same nature. A clonus quite similar may sometimes be obtained in other muscles—peronei, flexor brevis pollicis. Similar contractions may be obtained in the muscles of the arms. Normally those most distinct are (1) in the biceps and supinator longus, obtained by a tap at the wrist, especially on the radius, to which the biceps tendon is attached; and (2) in the triceps, by a tap on its tendon above the olecranon. When excessive, and there is persistent contraction in the flexors of the fingers, a similar contraction can be obtained in them, or in the supinators, and even a clonus, by maintaining the increased tension. With the increased excitability of disease, a contraction can easily be obtained in the masseters by tapping the chin when the jaw is depressed, and a clonus may occasionally be produced in these muscles, and even in the trapezius.

When a tendon is tapped, and its muscle contracts, the occurrence has so much the aspect of a true reflex action that it was generally assumed to be such, the stimulus being the excitation of nerves in the tendon. This view received apparent confirmation by the discovery of certain facts. (1) That there are nerves in tendon. (2) That these phenomena depend for their occurrence on the integrity of the reflex path to, through, and from the spinal cord, and are arrested by a lesion in this path. By experiments on animals (in whom similar contractions may be obtained) it has been found that they are prevented by division of the nerves to the muscles, by division of either the anterior or posterior roots of the spinal nerves, or by destruction of the spinal cord.†

These experimental facts are abundantly confirmed by those of tapped, the resulting contraction occurs in about ·033 sec.[*] The interval for the knee-jerk has been found by most to be about ·04 or ·033 or ·022 sec.[†] Gotch found it to be ·025 sec., and that between the tap on the tendon and a true reflex contraction of the opposite quadriceps to be ·11 sec.[‡]

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* Other illustrations will be found in the writer's paper ('Med.-Chir. Trans.,' 1879), "The so-called Tendon-reflex Phenomena." Since then tracings have been published by Charcot and many others.

† See Tschirjew, 'Archiv für Psychiatrie,' Bd. viii, Heft 3, 1878, and many subsequent investigations, among which the most important are those of Waller ('Journ. of Phys.,' 1896) and Sherrington ('Proc. Roy. Soc.,' 1893). They do not involve practical questions, such as to make their description needful here.

But it is certain that some reflex process is concerned in the phenomena. We have still to account for the irritability which permits the local stimulus to cause a contraction. This irritability is developed by passive tension. If the muscle is relaxed, the fibres may contract if they are struck directly, just as do the fibres of a separated frog's muscle, but no contraction can be produced by striking the tendon. Hence we must assume that the tension excites, by a reflex influence, a state of irritability to local mechanical stimulation,—such as that of a tap on the muscle, on its tendon, or even the vibration from a tap on the bone or on adjacent parts. But only that form of mechanical stimulation is effective which *suddenly* increases the previous tension. It is only because the tap on the tendon does this so readily that the tendon is the means by which the contractions are most easily produced, and through which they have been chiefly studied and prematurely named. If the tension put on a muscle is gentle and gradual, it may only develop the irritability, and an additional local stimulation is necessary to produce a visible contraction. If, however, the tension is sudden and forcible, it not only develops the irritability, but produces visible contraction in the muscle thus rendered irritable—as in setting up the foot-clonus. I have shown that the relaxation of the muscle, between the successive contractions, is not complete: there is persistent residual contraction, *i. e.* a tonic contraction on which the clonic contractions occur. When one clonic contraction is over, the tension continuing, a second is instantly developed.

The production of the clonus seems thus to depend on the limited duration of single muscular contractions (such as are obtained by a single induction shock[§]), the effect of sudden tension being exerted on all the fibres of a muscle at the same time. In the state of excitability produced by the moderate tension its effect on the relaxing fibres is

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[*] 'Med.-Chir. Trans.,' 1879, p. 292. An erroneous distinction between the nature of the knee-jerk and foot-clonus was made in this paper.

[†] ·039 sec., Burckhardt; ·032—·034 sec., Tschirjew; ·04 sec., Brissaud; ·03—·04 sec., Waller; ·03 sec., Eulenberg. Some of my own measurements ('Med.-Chir. Trans.,' 1879, p. 275) gave a longer interval when the movement of the foot was taken as the indication of the commencing contraction. "Load" will increase greatly the period of latent stimulation, probably by causing the initial contraction to expend itself on the elasticity of the muscle. The measurements given above were obtained by recording the commencing contraction of the muscle.

[‡] 'Journ. of Phys.,' 1896, vol. xx, p. 322. In the rabbit he found the interval for the tendon-contraction to be only ·005 sec., and compares this with Tigerstedt's measurement of the interval when the nerve is stimulated (·01 sec., twice as great), and concludes that the contraction can only be direct.

[§] See Foster's 'Physiology,' 1888, pt. i, p. 78.

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[illegible] dependent on a muscle-reflex
[illegible] ('Virchow's Archiv,'
[illegible] advocated by Gotch as th
[illegible] and is indeed now ge
[illegible] Cord,' 1883, p. 31;

bility.[*] The relation of these contractions to the nerve-centres will be considered in connection with these.

Paradoxical Muscular Contraction.—This name has been given by Westphal[†] to a slow tonic contraction occurring in a muscle when its attachments are suddenly brought nearer in some morbid states. It is best seen in the tibialis anticus; if the foot is grasped, and passively flexed on the leg, the tibialis anticus contracts, sometimes after an appreciable interval; its tendon stands out, and the contraction keeps the foot flexed for a time, which may be some minutes (in one case for twenty-seven): the relaxation is slow. Repetition lessens the contraction. The contraction is not voluntary (although a voluntary contraction may simulate it). It may occur on one side only. The contraction produced by brief faradism may be similarly prolonged in the same case. A similar contraction may be observed in the extensors of the toes, sometimes in the flexors of the knee, rarely in the arm muscles. It seems to be an excessive manifestation of a physiological relation by which suddenly diminished tension increases as increased tension lessens the activity of the motor centres. Tension stimulates the afferent nerves, and the relaxation of the opponents of a contracting muscle during movement seems to be thus produced, as is made probable by the absence of descent of the upper lid in palsy of the inferior rectus.[‡] Sherrington has shown that if the nerve to a muscle is cut and its central end stimulated the opponents relax.[§] Conversely we can understand that diminished tension may induce over-action. It seems to be an excess of the contraction by which a muscle adapts itself to passive shortening of its course. In the cases that have presented this phenomenon there has been no muscular rigidity, and no great excess (even sometimes a loss) of the myotatic irritability. It may occur in the early stage of tabes, and in some other affections, but its significance is not known. A phenomenon somewhat similar occurs in hysteria (Féré); it seems, indeed, but an isolated and imperfect instance of the condition that, when universal and perfect, constitutes the *flexibilitas cerea* of catalepsy, *i. e.* an unrestrained spinal reflex process in the centres concerned in the states of the muscles.

CHANGES IN NUTRITION.—The nutrition of all the tissue elements is largely under the influence of the nervous system. Whether this influence is exerted through special "trophic" nerves, or through the motor, the sensory, and especially through the vaso-motor nerves, is a question that has been much discussed in the past. The balance of evidence is against the existence of special trophic nerves or centres. The fact regarding these changes that is clearest and most important is that acute disturbance of nutrition is the result of irritative changes in the nutrition of the nerves, and is in proportion to the intensity of that irritation. If a thread is passed through each sciatic nerve, and one is also irritated by the application, from time to time, of irritant liquids, trophic changes occur in that limb with greater rapidity and far greater intensity than in the other (Lewaschew).

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[*] Risien Russell ('Proc. Roy. Soc.,' vol. liii, p. 430).

[†] 'Arch. f. Psych.,' Bd. x, p. 243.

[‡] See the Author, 'Med.-Chir. Trans.,' 1879.

[§] 'Brit. Med. Journ.,' 1893.

The same fact is frequently indicated by the extreme readiness with which trophic lesions of the skin, and even cellular tissue, occur in certain forms of inflammation of the spinal cord, especially in those that have a strong tendency to spread in the cord, and to the nerves, if they reach the centres of them. The only apparent explanation of the various facts is that the nerve-endings are related to the tissues in such a way that the nutrition of the molecules of the tissues is determined in its character by that of the nerves. The process of irritation descends the nerves; it passes from them to the tissues, and extends to all that are in continuity. Thus we can understand equally the acute changes in the muscles, which will be described as resulting from disease of the motor nerves, and also those in the skin and other tissues that are under the influence of the sensory fibres.

Nutritive changes in the skin are easily recognised, but they differ much in their character according to their acuteness. When they are rapid, and due to very acute irritation of the nerves or the spinal cord, such as those just mentioned, the temperature of the limb is raised, the vessels readily dilate, and remain dilated for a long time, bullæ form, containing a dark-coloured liquid, and slight pressure occasions a slough. These changes sometimes seem to occur spontaneously, but are more often excited by some cutaneous irritation, and very slight irritation of the skin will suffice to produce them. Trifling pressure will set up a slough, and extensive vesication may result from the application of a hot-water bottle that is not more than pleasantly warm to a healthy hand. When the nerve irritation is intense, effusion may occur into the joints.

In the case of the fifth nerve, acute trophic changes occur in the eyeball, chiefly when the disease involves the Gasserian ganglion, or the nerve in front of it. To produce the same effect, a lesion behind the ganglion has to be more irritating than one in front of it, and a similar relation obtains in the case of the spinal nerves and the spinal ganglia. The cells of the ganglia govern the nutrition of their processes in each direction, *i.e.* of the whole neuron, and we can understand that they have some power of hindering the propagation of irritative changes, while, if these invade the ganglion, they pass thence with special energy. Very intense changes in nutrition may thus pass to the skin in acute inflammation of the spinal cord.

The alterations in nutrition in chronic lesions (which have been carefully studied by Paget and Weir Mitchell) differ considerably from those that result from acute irritation. There is a slow change in the nutrition of the skin, which becomes red, thin, and shiny—the " glossy skin" of Paget. The subcutaneous tissue also wastes, so that the finger tips become pointed. The growth of the hair and nails is altered, and the latter become brittle. The bones may suffer in their nutrition, and may break more easily than in health. If the lesion occurs during the period of growth, this is retarded.* These changes seem to depend on slower change in the nutrition and vitality of the tissue elements corresponding to a slower change in that of the nerves, an atrophy rather than a degeneration.†

The *muscles* suffer from lesions of the motor nerves; the minute changes are described with the latter. At first there may be merely flabbiness, and an appearance of wasting that is not confirmed by measurement, but there is soon an actual diminution in the circumference of the limb. In extreme cases all the muscular tissue disappears, and the contour of the limb is considerably changed. In comparing the size of the limbs on the two sides it should be remembered that the limbs on the right side are normally somewhat larger than those on the left side, and that the difference varies according to the occupation of the individual, and the degree in which this involves a greater use of the limbs on one side. In measuring, great care is necessary to secure, as nearly as possible, the same conditions on each side, both as regards the state of the muscles and the place of measurement. It is best, wherever possible, to take the maximum measurement in each part, rather than to attempt to make the measurement in the same place. In the calf the maximum circumference should always be taken. In the thigh accurate measurement is extremely difficult, because a maximum cannot be taken. We may endeavour to measure at the same point in the thigh at the same distance from the condyles, but it is very difficult to be exact, and a more accurate comparison can often be made by taking the minimum circumference above the knee, in spite of the fact that the muscular tissue there is small, and the difference less than it is elsewhere. In the forearm the best result is obtained by taking the maximum measurement around the muscular prominence below the elbow, over the supinator longus. In the upper arm, the circumference is nearly the same in the middle third, and the measurement may be made halfway with little risk of error. In all cases care must be taken to draw the tape equally tight at each place. It is easier to do this with a flexible steel measure than with an ordinary tape measure. Exactness is best secured by having a spring at one extremity of the tape with an index that will show the tension.

ELECTRICAL IRRITABILITY.—The nerves and muscles are excitable by electricity, and the excitability is changed by disease, of which the change is often an important symptom. It indicates the state of nutrition of the nerve-fibres and muscles, and from this we can often draw important inferences regarding the condition of the centres.

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* But the subject of hindrance to growth is complex, and still not clear. See the account of infantile paralysis.

† Cf. Marinesco on degeneration and atrophy ("La Theorie des Neurones," 'Presse Méd.,' 1895).

In the normal state nerve-fibres are stimulated by either the induced or the voltaic current, the stimulation of the motor nerves being shown by contraction in the muscles supplied by them, that of the sensory nerves by the sensation that is caused. The contraction of the muscles is continuous when the faradic current is applied, but if the isolated shocks of which the current consists are separately passed, each causes a brief, momentary contraction. When the voltaic current is applied, contraction occurs, with a current of moderate strength, only when the strength of the current is changed, and chiefly when the current commences or ceases to pass, *i. e.* when the circuit is "made" or "broken." The stimulation of the sensory nerves is greatest at those times, but occurs also, in a slighter degree, during the whole time that the voltaic current is passing. Hence this is probably the case also in the motor nerves, although the stimulation is too feeble to produce a contraction in health with a strength of current that can be borne. In proportion as the nutrition of the nerve-fibres is impaired, their excitability is lowered, and a stronger current of each kind is required to excite them and cause contraction in the muscles they supply. When their nutrition is much impaired—*i. e.* when the fibres are "degenerated"—no contraction can be obtained even with the strongest currents.

The changes in the excitability of the muscles are less simple, because in them there are two excitable structures—the terminal branches of the nerves, and the muscular fibres themselves. Of these the nerve-fibres are the more sensitive to faradism, and the faradic stimulation of a muscle, under normal circumstances, is by means of these motor nerve-endings. Thus we find that its excitability corresponds in degree to that of the motor nerve supplying it. The muscular fibres themselves are, even in the normal state, less sensitive to faradism than the nerve, apparently because they are incapable of ready response to a stimulus so very short in duration as are the shocks of which the faradic "current" consists. The proof of this consists in the fact that under the influence of curara, which removes the excitability of the terminations of the motor nerves, the muscle requires a stronger faradic current to stimulate it than in the normal state. But under the influence of curara, or when the nerve is degenerated, the slowly interrupted voltaic current stimulates the muscle as readily as in the normal state; a contraction occurs when the circuit is completed or broken—slower than that which occurs when the nerve-fibres are intact, and due to the stimulation of the protoplasm of the muscular fibres themselves. The fact that, under normal circumstances, the contraction which is caused by the voltaic current is as quick as that produced by the faradic shock, is ground for believing that, in health, the voltaic as well as the faradic current causes the muscle to contract by exciting the motor nerve-endings. When the motor nerve is degenerated, and will not respond to faradic or voltaic stimulation, the application of the former to the muscle ceases to cause contraction. Apparently, the nerve-degeneration is accompanied by changes in the nutrition of the muscular fibre, by which any power of response to faradism, which it possessed in the normal state, is lost. But not only does the response to the voltaic current remain; it becomes more ready than in health, doubtless in consequence of nutritive changes just mentioned. Moreover there may often be observed a change in the readiness of response to the two poles of the voltaic current—a "qualitative" change, as it is termed. In health, the first contraction to occur, on gradually increasing the strength of the current, is at the negative pole when the circuit is closed, and a stronger current is required before closure-contraction occurs at the positive pole. But, in the morbid state we are considering, closure-contraction may occur at the positive pole as readily as at the negative, or even more readily,—and contractions, when the circuit is broken, occur far more readily than in the normal state. This condition, then—faradic irritability lost, voltaic irritability increased and often changed in quality—is termed the "degenerative reaction," because it occurs when the nerve-fibres are degenerated; if we test *them* we shall find no response to any stimulus, voltaic or faradic. It thus indicates loss of excitability in the motor nerves within the muscles, or a change in their endings on the fibres, by which these cannot be excited, either by electricity or by nerve-impulses, in the fibres. It occurs when the latter has been produced by some toxic agent, or the fibres also are degenerated in consequence of their damage between the muscle and the ganglion cells of the cord of which they are part, or destruction of the cells.

But the motor nerve-cells and fibres often undergo changes in nutrition of a much more chronic character. In this condition the excitability of the fibres is lessened gradually and slowly. The irritability of the intra-muscular nerve-endings is lowered in the same degree as that of the nerve-trunks, and we have a similar diminution to both faradism and voltaism. The nutrition of the muscular fibres is slowly, gradually impaired; and when the nerve-fibres are much affected the muscular fibres are also. There is no stage in which the nerve-fibre excitability is lost, and the muscle-fibre excitability retained; hence there is no condition of lost faradic and increased voltaic excitability such as characterises the degenerative reaction just described. Excitability is changed to the one form of stimulus just as to the other.

Between these two forms there are intermediate conditions. For instance, the nerves may present normal irritability, while in the muscle there is often increased voltaic excitability and a changed order of polar reaction. In these cases some nerve-fibres are degenerated, and lead to the increased excitability of some muscular fibres. In both nerve and muscle the character of the reaction is manifested by the more excitable structures; hence it is normal in the nerve and altered in the muscle—this has been termed by Erb the "middle form of degenerative reaction." It is more accurate to call it the "mixed form."

Another common intermediate form is due to the degeneration of the nerves, although slow, being less slow than in the condition described above. Hence the nutrition of the muscular tissue and its excitability (by voltaism) persist longer than those of the nerves, and although lowered, are diminished less than the faradic excitability of the nerves at each stage. When the latter is lost, voltaism will still cause a slight contraction in the muscles, which may occur first at the anode instead of the cathode. Every gradation, moreover, is met with between this and the true reaction of degeneration. This will be intelligible on consideration. It depends chiefly on the rate at which the nerves degenerate.

The lowered excitability of the nerves, due to degeneration, is often preceded by a slight increase of irritability, very transient when the degeneration is acute, of longer duration when the degeneration is of the slower variety just noticed. In some morbid states, again, in which the change of nutrition in the cells and fibres is extremely slight, an increase may alone be discovered. I have found such an increase, for instance, in diseases regarded as functional, as paralysis agitans and chorea, and it is an interesting proof of the molecular changes which underlie, or result from, so-called "functional" maladies.

The various changes in irritability were formerly thought to indicate the existence, and various affection, of separate centres for the nutrition of the nerves and muscles, apart from, though acting through, the motor nerve-cells. Remembering that the fibres of the nerves and muscles suffer in different degrees, as above described, the phenomena may all be explained on the simpler principle stated, without the assumption of these special centres, of the existence of which there is, indeed, no evidence. Special trophic nerves and centres for muscular nutrition have quietly disappeared from physiology, and are now only matters of interest to the student of its history, and to those whose memories reach further than two decades.

It is, however, important to remember that such alterations in nutrition and excitability as occur in the whole course of the motor fibres when the cells are affected (of which they are the prolonged processes) may have a different origin at the periphery. They are the same in "degenerative peripheral neuritis," which begins in the extremities of the fibres (where vital resistance is least in consequence of distance from the cell body), and extend for a variable distance upwards. But this condition is seldom, if ever, so slow as to permit the equal loss of nerve and muscle excitability met with in the central degenerations.

THE MUSCLES: THEIR ACTION AND PARALYSIS.

Disease of the motor nervous system is largely manifested by loss of muscular action. Individual muscles, as well as groups of muscles, are often separately affected. Hence it is desirable to consider the symptoms of the paralysis of the more important muscles, before we enter on the study of special diseases.

The symptom of the palsy of any muscle is a loss of its normal action, and a knowledge of this action is essential for the comprehension of those symptoms. The two must therefore be considered together. They are positive and negative aspects of the same facts. It may be well, at the same time, to mention the nerve by which each muscle is supplied, and also the spinal roots from which the fibres come. These cannot be ascertained by dissection. Of the several roots that join to form the cord of the plexus from which the nerves to two muscles come, fibres from all may pass into one nerve, and from only one or two of the roots into another. The facts have been learned from experiment and the effects of disease and injury. Their practical importance is great.

The action of muscles is threefold. (1) By their tonic contraction they maintain the parts in a certain posture, independently of voluntary effort. By actual contraction they (2) produce certain movements, and also (3) oppose the action of other muscles by a feebler contraction, and thus steady the movement that results.

The complex way in which muscles act together, and modify each other's effect, renders the subject a very large one. Here only an outline can be given of the more salient facts concerning the most important muscles. The reader who desires to pursue the subject further can do so in the 'Physiologie des Mouvements' of Duchenne, whose investigations by means of faradism and his observations on disease were so careful and so extensive as to leave little but confirmation to subsequent workers.

In the following account the nerve that gives the branch to the muscle is first given, and then the number of the nerve-root or roots—(C)ervical, (D)orsal, or (L)umbar—from which the nerve-fibres come.

Special Thoracic Muscles.

The **Diaphragm** (C. 4 chiefly, phrenic nerves), although a double muscle with two nerves, habitually acts as a whole, the two halves contracting simultaneously and diminishing each lateral curve of the arch. The central tendon descends but little. The abdominal viscera are depressed, and the parietes protruded. If the hand is placed beneath the ribs, the descent of the viscera beneath the diaphragm can be felt. When the diaphragm contracts alone, as when the intercostals are paralysed, or the phrenic nerve is faradised, the ribs to which change is often an important symptom. It indicates the state of nutrition of the nerve-fibres and muscles, and from this we can often draw important inferences regarding the condition of the centres.

In the normal state nerve-fibres are stimulated by either the induced or the voltaic current, the stimulation of the motor nerves being shown by contraction in the muscles supplied by them, that of the sensory nerves by the sensation that is caused. The contraction of the muscles is continuous when the faradic current is applied, but if the isolated shocks of which the current consists are separately passed, each causes a brief, momentary contraction. When the voltaic current is applied, contraction occurs, with a current of moderate strength, only when the strength of the current is changed, and chiefly when the current commences or ceases to pass, *i. e.* when the circuit is "made" or "broken." The stimulation of the sensory nerves is greatest at those times, but occurs also, in a slighter degree, during the whole time that the voltaic current is passing. Hence this is probably the case also in the motor nerves, although the stimulation is too feeble to produce a contraction in health with a strength of current that can be borne. In proportion as the nutrition of the nerve-fibres is impaired, their excitability is lowered, and a stronger current of each kind is required to excite them and cause contraction in the muscles they supply. When their nutrition is much impaired—*i. e.* when the fibres are "degenerated"—no contraction can be obtained even with the strongest currents.

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But the motor nerve-cells and fibres often undergo changes in nutrition of a much more chronic character. In this condition the excitability of the fibres is lessened gradually and slowly. The irritability of the intra-muscular nerve-endings is lowered in the same degree as that of the nerve-trunks, and we have a similar diminution to both faradism and voltaism. The nutrition of the muscular fibres is slowly, gradually impaired; and when the nerve-fibres are much affected the muscular fibres are also. There is no stage in which the nerve-fibre excitability is lost, and the muscle-fibre excitability retained; hence there is no condition of lost faradic and increased voltaic excitability such as characterises the degenerative reaction just described. Excitability is changed to the one form of stimulus just as to the other.

Between these two forms there are intermediate conditions. For instance, the nerves may present normal irritability, while in the muscle there is often increased voltaic excitability and a changed order of polar reaction. In these cases some nerve-fibres are degenerated, and lead to the increased excitability of some muscular fibres. In both nerve and muscle the character of the reaction is manifested

the muscle is att
causes a slight
is lost in the action
of the upper part
instead of advancing.
There often result
and abdomen, retra

STERNO-MASTOID
cervical plexus). pa
the mastoid process,
side on which the m
the vertical position,
beyond this position.
of the head, and but
loss. There is no such t
muscles the head can be
can be brought forwar
associated in action with
muscle." For instance,
by the left sterno-mastoid.

MUSCL

MUSCLES MOVING TH
spinal accessory (C. 2 to 6,
consists of three parts. T
the clavicle, is rarely used

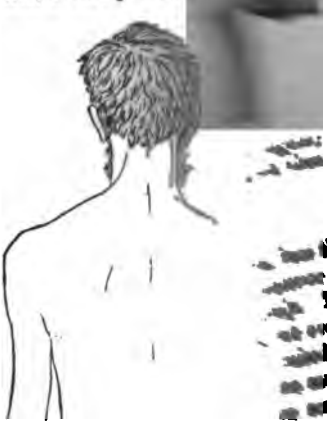

FIG. 5.—Paralysis and wasti
pezius; alteration in
shoulder at rest.

The second part is that which
upper three dorsal spines, dow

lower angle in) from the
is moved forwards by the
held against the thorax and
its vertical axis by the action of
and of the middle part on the
the thorax, leaving a groove
Fig. 8). The scapula is at the
upwards. Elevation of the arm
weakened, but can be imperfectly
Loss of the serratus weakens
inspiratory expansion of the thorax.
the paralysed side (Poore).

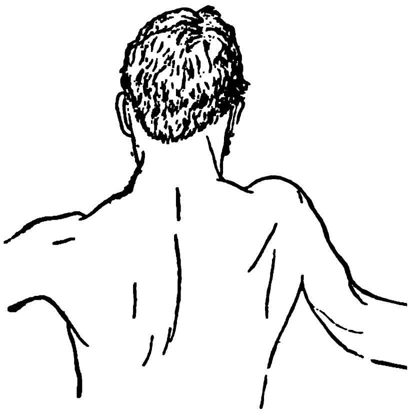

FIG. 9.—Paralysis of right deltoi
vation of shoulder by trapezius
attempt to raise the arm, w
slightly abducted by the supras

the brachial plexus posterior cor
and posterior fibres also moving
The arm is raised least by the p
the latter only elevate it t
by the anterior fibres, and the
time depressed. Elevatio
scapula (trapezius and serratus)
deltoid, preventing the rotation
alone would cause. In
backwards, is almost lost
ement by the supraspi
the scapula and elevat
of the associated tr
when the deltoid
the corresponding
lysed, there is sti

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* Anatomists now believe tha
muscle, and that it is wholly supp
fact that its action is chiefly with

power of direct abduction by the conjoined contraction of the anterior and posterior parts, aided by the supraspinatus.

The *Supraspinatus* (suprascapular nerve—C. 4 and 5) abducts the arm, moves it forwards, and rotates it in. It thus aids the deltoid. Isolated paralysis of the supraspinatus has little influence on movement or position; but if the deltoid is also paralysed, the head of the humerus falls away from the acromion far more than when the deltoid is paralysed alone.

The *Infraspinatus* (suprascapular nerve—C. 4 and 5) rotates the humerus outwards, and in paralysis this movement is lost. A difficulty in writing is produced, the movement along the line being by this rotation of the humerus.

The *Teres minor* (circumflex nerve—C. 5) has a similar action to the infraspinatus, and its palsy has a similar effect.

The *Subscapularis* (short subscapular nerve, from the fifth and sixth cervical) rotates the humerus in, and its paralysis lessens this movement.

The *Latissimus dorsi* (long subscapular nerve, from the brachial plexus, posterior cord—C. 7) lowers the raised arm, and puts it back; the upper part adducts the scapula, the lower depresses the shoulder by acting on the humerus, which it tends to drag out of the socket. It inclines the trunk a little, and both muscles together extend the trunk. In paralysis, forcible backward depression of the raised arm is lost, and the shoulder cannot be put back without being also raised (by the trapezius).

The *Pectoralis major* (anterior thoracic nerves from the brachial plexus, outer and inner cords—C. 5, 6, and 7) consists of two muscles, the action of the clavicular and sternal parts being different. The clavicular (which arises also from the highest part of the sternum), if the arm is hanging, brings the shoulder forwards and upwards, as if shivering; if the arm is raised, it is brought forwards and lowered to the horizontal position. The muscle is thus concerned, Duchenne says, in the "cut" of the swordsman and the benediction of the priest. The sternal portion lowers the raised arm from every position, and if the arm is hanging, it draws the shoulder down. Paralysis of the upper part has little effect on the movement of the arm, because the anterior fibres of the deltoid have the same action. It is easily recognised by making the patient put his arms in front of him and press the palms together. In paralysis of the lower part, even with the latissimus, the raised arm can still be lowered accurately by the weight of the arm and relaxation of the elevators, but it cannot be lowered against even a slight resistance. Thus a blacksmith with this defect could wield a heavy hammer, but could not blow the bellows by pulling downwards a cord (Duchenne).

The *Teres major* (short subscapular nerve from the brachial plexus posterior cord—C. 7) approximates the humerus and the outer edge of the scapula, by bringing the former to the side of the trunk, and rotating the latter. The simultaneous contraction of the levator anguli and rhomboids, fixing the inner angle, causes this rotation to raise the prominence of the shoulder. Hence, in a forcible elevation of the shoulder, the arm is pressed against the side, the lower fibres of the latissimus and pectoralis major aiding the adduction. The teres cannot alone put the arm behind the trunk. In paralysis, the elevation of the shoulder, with the arm against the side, is lost.

MUSCLES MOVING THE FOREARM.—*Triceps* (musculo-spiral nerve—C. 6 and 7).—The long head has an action similar to the teres, but feebler. It contracts when the arm is forcibly lowered, and prevents the displacement downwards of the head of the humerus by the actual depressors, the latissimus and pectoralis. All parts extend the elbow, the long head with less force than the others, but its action on the shoulder-joint, just mentioned, is important, because forcible depression of the raised arm is often associated with extension of the elbow. In paralysis of the triceps the elbow can only be extended by the weight of the forearm, and extension against gravitation is impossible. Thus a man with paralysis of the triceps cannot raise his hat in the customary manner. Flexion of the elbow is uncertain, on account of the loss of the antagonistic steadying force.

The *Brachialis anticus* (musculo-cutaneous and musculo-spiral nerves) flexes

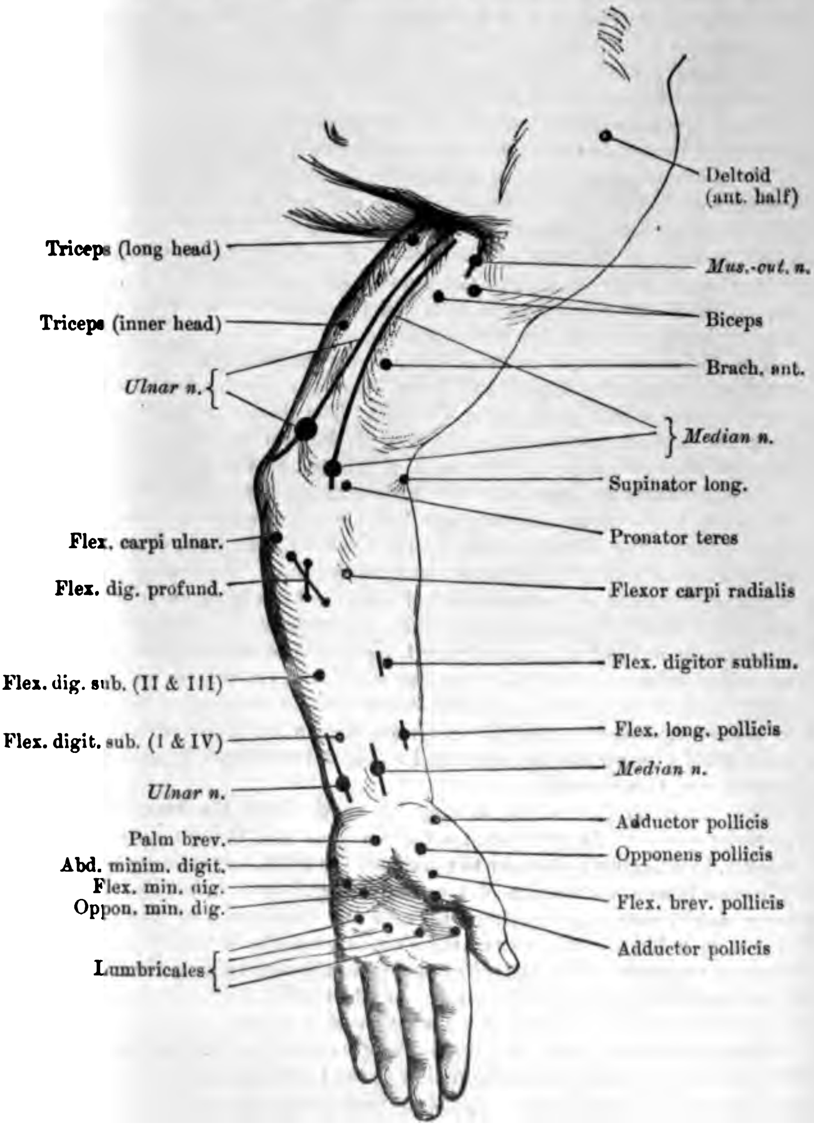

FIG. 10.—**Motor points for the arm, inner side (from Erb). The points at which the muscles and nerves can most effectively be stimulated.**

the elbow simply. Its rare isolated palsy has little effect, since it is supplemented by the biceps and supinator longus.

The *Biceps* (musculo-cutaneous nerve—C. 4, 5, and 6) supinates the forearm if it is pronated, and then flexes the elbow. In paralysis the flexion can still be effected, but the traction on the humerus causes pain at the shoulder, from the loss of the support of the long head of the biceps.

The *Supinator longus* (musculo-spiral nerve—C. 4 and 5) places the forearm midway between pronation and supination, and then flexes the elbow. If it is paralysed there is a tendency for supination to accompany flexion (biceps); and if the brachialis anticus is also paralysed, the elbow can only be flexed when the forearm is supinated.

If the three direct flexors of the elbow are paralysed, feeble flexion is still possible by the extensors of the wrist, which cross the elbow-joint, but only after the forearm has been pronated and the wrist over-extended.

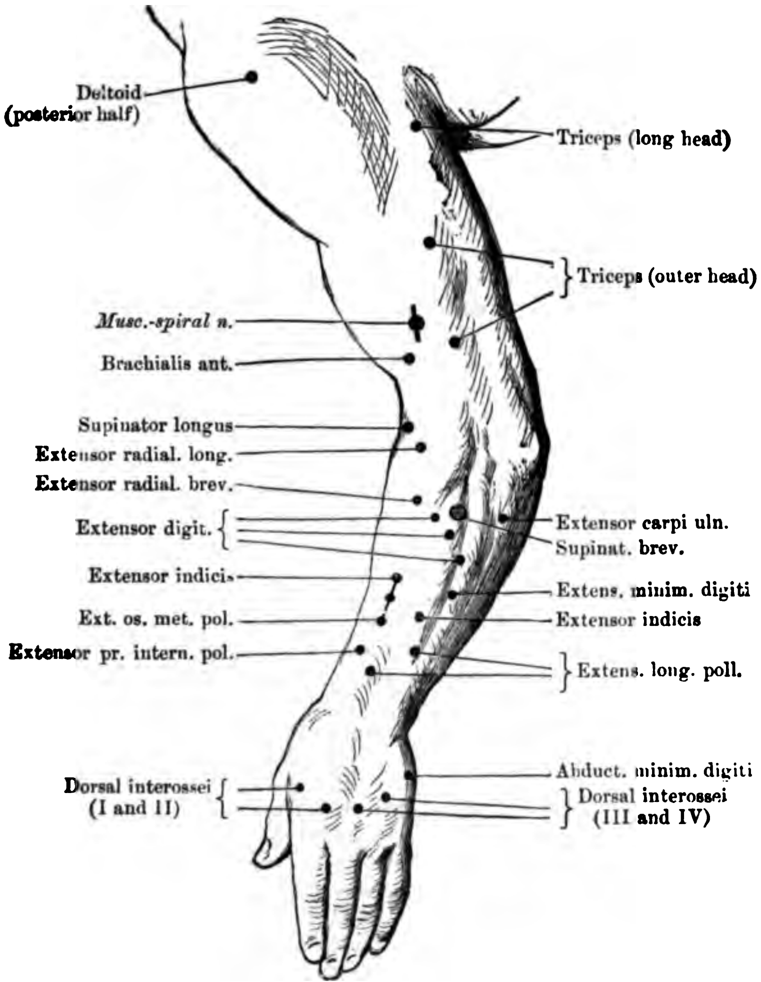

FIG. 11.—Motor points for the arm, outer side (Erb).

The *Supinator brevis* (musculo-spiral nerve by posterior interosseous branch—C. 5) is the only simple supinator. If it is paralysed, supination can still be effected by the biceps, and also by the supinator longus as far as midway between pronation and supination.

Pronators.—The *p. teres* and *p. quadratus* (median nerve—C. 6 and 7) both pronate strongly, and their palsy causes loss of this movement, but pronation to the mid-position is still possible by the supinator longus.

MUSCLES MOVING THE HAND.—*Flexors of Wrist.*—The *F. carpi ulnaris* (ulnar nerve) and *F. carpi radialis* (median—C. 7 and 8) flex the wrist. The ulnar flexor tends to turn the supinated hand still more out, but neither moves the wrist-joint laterally. Flexion indeed hinders the lateral movements of the wrist, in consequence of the shape of the articular surfaces. The f. c. ulnaris flexes the fifth metacarpal bone on the carpus as well as the wrist-joint. In paralysis, flexion of the wrist can only be effected by the flexors of the fingers when these are extended. Extension of the wrist is unsteady from the loss of the antergic contraction of the flexors (see p. 10).

Extensors of Wrist.—*Extensor carpi radialis brevis et longus ; E. c. ulnaris* (musculo-spiral nerve and its posterior interosseous branch—C. 6 and 7). The short radial is a direct extensor; the long radial and the ulnar move the hand laterally as well. In paralysis of all three extensors the wrist can only be extended by the extensors of the fingers when the phalanges are flexed. In loss of the short radial, direct extension is still possible by the long radial and ulnar, and lateral extension by one of these alone. If either is paralysed, together with the short radial, direct extension is lost, and only lateral extension, in the direction of the remaining muscle, is possible. Paralysis of either lateral extensor, long radial or ulnar, leads to permanent deviation of the wrist in the direction of the remaining muscle (Fig. 12). The loss of the long radial is more serious than that of the ulnar, because the radial lateral movement is of more importance, being needed for the convenient motion of the hand to the mouth. Paralysis of the extensor impairs flexion of the fingers, from the great shortening of the course of the tendons by the flexion of the wrist that occurs. The ulnar extensor acts also synergically with the extensor of the metacarpal bone of the thumb, as may be noted if the finger is placed on the tendon beneath the styloid process of the ulna. Hence, in paralysis of this extensor, the hand deviates laterally when the thumb is strongly extended.

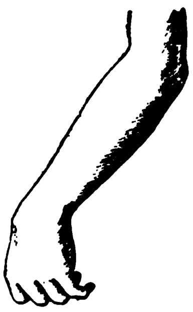

FIG. 12.—Paralysis of the long radial extensor of the wrist in a young child; habitual deviation of the hand towards the ulnar side. (After Duchenne.)

Extensors of the Fingers.—*Extensor communis digitorum ; E. indicis ; E. minimi digiti* (musculo-spiral nerve—C. 6 and 7). The common extensor moves the fingers and then the wrist. When the muscle is faradised, the extension begins at the distal phalanges, and these become flexed again, when the hand is extended beyond the plane of the forearm, by the tonic force of the flexors, the course of their tendons being elongated by the extension of the wrist. Moreover the muscle has little action on the last two phalanges, since they cannot be extended by the long extensor, if the interossei, their proper extensors, are paralysed. During extension by the communis the fingers are separated from the second. The extensors of the first and last fingers have a similar extensor action, but, in addition, they adduct their respective fingers towards the middle finger. In paralysis, the extension of the fingers is impossible; but if the proximal phalanges are passively extended, the middle and distal joints can be extended by the interossei. For the lateral movements of the digits, extension of the proximal phalanges is essential, and hence these movements are lost, but they can be performed if the proximal phalanges are passively extended. The posture of the fingers due to contraction of the palmar fascia resembles that in palsy of the long extensor (see Fig. 13), but an examination of the palm shows the cause of the flexion.

Flexors of Fingers.—*F. sublimis* (median nerve—all fibres C. 7 and 8); *F. profundus* (median and ulnar nerves). These muscles flex chiefly the second and third phalanges, the first phalanx being flexed by the interossei. The superficial muscle flexes the second phalanx on the first, the deep flexes both. The action on the first phalanx is confined to extreme flexion of the fingers, and is the less, the more the wrist is flexed. But if the flexion of the middle and distal phalanges is prevented the first is strongly flexed. In extreme shortening of the course of the tendons by flexion of the wrist, the action on the fingers is very feeble, evidence of a normal antergic action of the extensors of the wrist.

FIG. 13

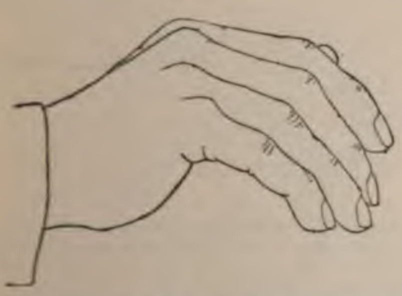

FIG. 14.

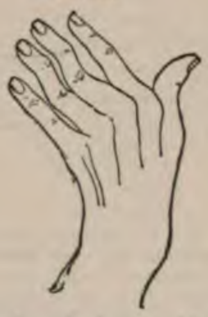

FIG. 13.—Posture of the hand in contraction of the palmar fascia, resembling that in paralysis of the long extensors of the finger.

FIG. 14.—Paralysis of the fibres of the flexor sublimis which act on the two middle fingers: twelve years' duration. The second phalanges of these fingers are bent backwards and subluxated from the contracture of the unopposed interossei, while the last phalanges are kept in position by the unaffected flexor profundus. (After Duchenne.)

When the extensor of the fingers is in strong action, extending the proximal phalanges, the action of the flexors on the second and third joints is very strong (tearing position). In paralysis of these muscles the power of flexing the last two joints is lost, but the interossei still flex the metacarpo-phalangeal joints. Paralysis of the deep flexor alone causes merely loss of the power of flexing the distal joint, but this impairs many movements, such as playing on the piano. In paralysis of these muscles, the unopposed tone of their opponents, the interossei, which extend these joints, leads in time to over-extension, and, with repeated passive pressure in using the fingers, may even produce a subluxation backwards. In palsy of the sublimis this effect is chiefly seen at the middle joint (Fig. 14), in that of the profundus at the distal joint.

Interossei and Lumbricales (ulnar nerve, except the outer two lumbricales, which are supplied by the median—C. 8 and D. 1).—The interossei abduct and adduct the fingers, but only when these are extended at the metacarpo-phalangeal joints, and some effort is required for adduction, since the tendency of the long extensor is to separate the fingers, and this influence has to be overcome. They also extend the second and third phalanges on the first, and flex the first on the metacarpal bones. The lumbricales aid the flexor-extensor action of the interossei, but do not move the fingers laterally. The opposite action of the forearm muscles and of the interosseous extensors and flexors is very important. Their synergic action steadies movements, and in many actions they contract alternately. Thus in making a down-stroke with a pen or pencil the long flexors bend the last two joints; while in making an up-stroke these are extended, and the metacarpo-phalangeal joint is flexed, by the interossei. In paralysis of these muscles the lateral movements are lost, but a slight abduction and adduction of the index can still be effected by its long extensors. Only the first phalanx can be extended, and flexion is almost confined to the last two phalanges. The first two lumbricales, being supplied by the median nerve, often escape when the other muscles are paralysed by an injury to the ulnar nerve, and they aid the others when these are merely weak; hence the index and middle fingers seem to recover before the others (Fig. 15). The position of

FIG. 15.

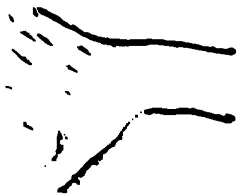

FIG. 16.

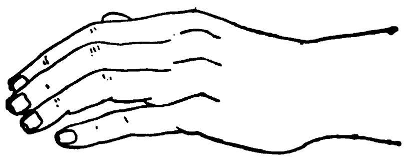

FIG. 15.—Incomplete paralysis of the interossei from a punctured [illegible] the ulnar nerve at the wrist: attempt to extend fingers. The loss [illegible] of the last two phalanges is chiefly marked in the third and fourth [illegible] from the influence of the lumbricales (supplied by the median) on the [illegible] (After Duchenne.)

FIG. 16.—Paralysis of the interossei (ulnar nerve) slight in degree: attitude [illegible] at rest.

[illegible] becomes altered. Normally there is slight flexion at all joints [illegible] of the muscles, interossei and long flexors. In paralysis the first [illegible] in a line with the metacarpal bones, while the other phalanges are [illegible] middle more than the distal (Fig. 16). In action this flexion is [illegible] the metacarpo-phalangeal joints become over-extended, and [illegible] strongly flexed (Fig. 17). Gradually the hand assumes this [illegible] (Fig. 18), and ultimately the posture becomes warped into [illegible] by the over-extension of the first phalanges, and extreme flexion of [illegible] by the contracture of the long extensor and of the flexors; the [illegible] muscles stand out conspicuously on the back and in the palm. [illegible] attitude is developed, the "main en griffe" (Figs. 19 and 20). [illegible] may ultimately limit even passive movement.

[illegible] *Extensor secundi internodii pollicis* (musculo-[illegible] interosseous branch—C. 8 and D. 1) extends both [illegible] the whole thumb backwards and from the fingers, so as [illegible] the plane of the metacarpus. It may ultimately extend the [illegible] It is not used in extending the thumb when this is opposed to the first finger. In paralysis, the metacarpal bone of the thumb is slightly flexed on the carpus, and is inclined forwards. The second phalanx is flexed on the first, and can only be extended (by the abductor and outer part of the short flexor) when the metacarpal bone is adducted and the first phalanx is flexed. The constant flexion of the second phalanx interferes with the movement of the index finger, unless the patient remembers to move the thumb out of the way by the extensor of the metacarpal bone. Writing is not interfered with because the muscle is not concerned in extension with opposition.

FIG. 17

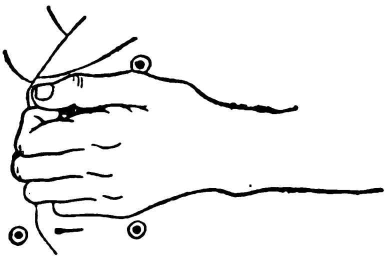

FIG. 18.

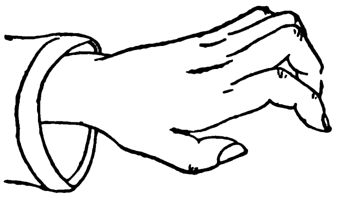

FIG. 19.

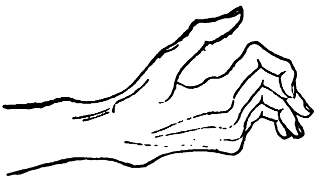

FIG. 20.

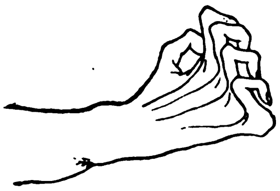

FIG. 17.—Attempt to unbutton waistcoat by the hand shown in the last figure; extreme flexion of the last two phalanges, and extension of the first, on the attempt to use the fingers. (From nature.)

FIG. 18.—Old-standing palsy of interossei and thenar muscles, showing the over-extension of the first and flexion of the last two phalanges.

FIG. 19.—Paralysis of all the intrinsic muscles of the hand and of the long flexor of the thumb, in consequence of an injury to the brachial plexus in dislocation of the shoulder. The fingers present the claw-like attitude; the thumb is in extension. (After Duchenne.)

FIG. 20.—Paralysis of the ulnar nerve from a wound at the wrist (indicated in the figure). Extreme claw-like hand from the unopposed contraction of the common extensor and long flexors of the fingers and thumb. (After Duchenne.)

The *Extensor primi internodii pollicis* (musculo-spiral nerve, post-inteross. branch—C. 8 and D. 1) is the true abductor of the thumb. It moves the metacarpal bone outwards, and extends the first phalanx. It would move the whole hand in the same direction as the thumb, were not this tendency counteracted by the antergic contraction of the extensor carpi ulnaris (q. v.). It does not pronate or supinate. In paralysis abduction of the metacarpal bone is less than normal. There is an undue flexion of the first phalanx, and the metacarpal bone is flexed on the carpus, so that the thumb is drawn towards the palm. The loss of this muscle is, however, compensated to a considerable extent by other muscles.

The *Extensor ossis metacarpi pollicis* (musculo-spiral nerve, post-inteross. branch—C. 8 and D. 1) is really the long abductor of the thumb. It moves the metacarpal bone outwards and forwards, flexing it on the carpus, and then flexes the wrist with slight pronation. It thus moves the thumb as much forwards as outwards. In *paralysis* the metacarpal bone is, at rest, less inclined forwards than normal, and somewhat abducted, but the first phalanx is in its normal position. Movement of the thumb is but little interfered with.

In combined palsy of the extensors of the first phalanx and of the metacarpal bone the thumb becomes adducted, and is parallel to the radius. The first phalanx is slightly flexed by the thenar muscles.

The *Thenar muscles* constitute two groups: (1) The short *abductor and outer portion of the short flexor* (median nerve—C. 8 and D. 1) move the metacarpal bone forwards and inwards (flexing the first phalanx), incline it outwards, and rotate it inwards, so as to place its palmar aspect opposite the fingers. The second phalanx is ultimately extended. If the metacarpal bone is previously abducted the movement is greater, and amounts to circumduction. (2) The *adductor and inner part of short flexor* (ulnar nerve—C. 8 and D. 1) go to the inner side of the first phalanx. The metacarpal bone is moved towards that of the second finger: if previously flexed, it is extended; if previously opposed to the index, it is moved a little outwards. The phalanges follow the movements of the metacarpal bone, but the first is slightly flexed and the second is extended, as the fingers are by the interossei.

The *Opponens pollicis* (median nerve—C. 8 and D. 1) flexes the metacarpal bone on the carpus, and abducts it, but this movement is insufficient to oppose the thumb to the index; the conjoint action of the abductor is necessary (see above).

FIG. 21.

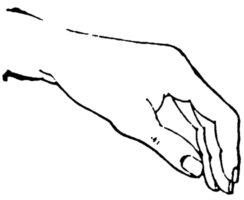

FIG. 22.

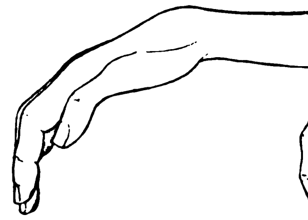

FIG. 23.

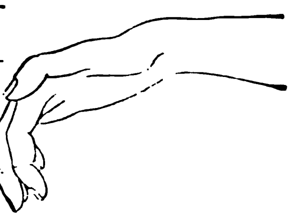

FIG. 21.—Normal position of the thumb (for comparison with the succeeding figures).

FIG. 22.—Position of the hand in long-standing paralysis and wasting of the thenar muscles. Under the influence of the long extensor the metacarpal bone of the thumb has been brought into the same position as the other metacarpal bones, being rotated slightly, so that the back of the thumb is in the plane of the back of the hand, like the hand of the ape. (After Duchenne.)

FIG. 23, from another case, shows the same condition, but still greater displacement of the metacarpal bone has taken place, from the greater contraction of the extensor. (After Duchenne.)

The *Flexor longus pollicis* (median nerve—C. 7 and 8, and D. 1) flexes the second phalanx forcibly and the first feebly. It has no action on the metacarpal bone. It is used in writing (making a stroke towards the body) and in picking up a small object, &c. In paralysis this flexion is lost, and with it these actions; if an object is held between the tips of the thumb and forefinger, the last phalanx of the thumb is bent back. Other movements of the thumb are not interfered with.

If all the thenar muscles are paralysed the metacarpal bone is in the plane of the index, and drawn towards it by the extensor of the second phalanx, which moves the metacarpal bone inwards and backwards. The whole thumb corresponds with the metacarpal bone in position, the phalanges being normal. In paralysis of the short abductor and flexor the second phalanx cannot be extended unless the metacarpal bone is abducted. These short muscles normally prevent the abduction that the extensor of the second phalanx tends to produce. The latter, moreover, prevents undue adduction when the special abductors are paralysed, but brings the metacarpal bone into the plane of the index. Thus the posture of the hand at rest resembles that of the hand of an ape (Figs. 21 and 22). If the abductor and opponens are paralysed, the tips of the thumb and fingers can only be brought together by flexing the last phalanges of the digits (Fig. 24). Then the thumb can be brought into contact with the finger by means of the short flexor, which inclines the metacarpal bone sufficiently to effect this, although not enough for the tip of the first finger to touch the thumb when its phalanges are extended.

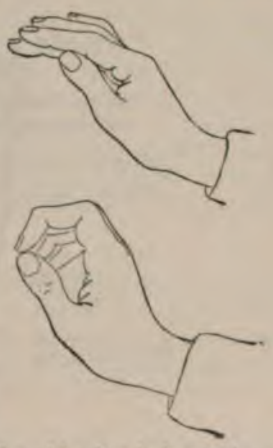

FIG. 24.—Paralysis of the abductor brevis and opponens pollicis. From the want of these muscles the thumb can only be brought in contact with the tip of the index by strong flexion of the last two phalanges of the fingers, otherwise the tip of the thumb only reaches the middle of the second phalanx. (After Duchenne.)

If the short flexor is paralysed, the thumb can still be opposed to the first two fingers by the abductor, but it cannot be opposed to the last two fingers on account of the deficient lateral inclination of the thumb, which should be produced by this muscle. Writing is easy by means of the short abductor, whereas if this is lost, although the thumb can be opposed to each of the fingers, writing is much interfered with.

If all the thenar muscles are paralysed, a certain amount of opposition of the thumb and fingers is still possible, by means of the flexion of the last phalanges of the thumb and fingers. If all are paralysed except the adductor, objects can still be held between the thumb and side of the palm.

MUSCLES OF THE LOWER LIMB.

MUSCLES MOVING THE HIP-JOINT.—The *Gluteus maximus* (inferior gluteal nerve, small sciatic, and a special branch from sacral plexus—L. 4 and 5, S. 1) extends the hip-joint, and freely rotates the thigh outwards. It is the most powerful extensor of the hip, and it is chiefly used when a forcible extension is required, and the joint has been previously flexed. It is employed, not in standing, or in walking on level ground, but in going upstairs, or uphill, and in rising from a seat When it is paralysed these movements are difficult.

The *Gluteus medius* (gluteal nerve—L. 4 and 5, S. 1) is the chief abductor. All parts of the muscle have this action, but in addition the anterior third moves the thigh forwards and rotates it inwards, while the posterior third moves it backwards and rotates outwards. The successive action of the several parts causes circumduction. The *Gluteus minimus* (gluteal nerve) has probably the same action. In paralysis, abduction and circumduction are lost; in standing on the other foot the pelvis is inclined on the affected side, and hence, in walking, there is an oscillation of the trunk, which becomes very considerable if the muscles of both sides are affected. Moreover the unopposed tone of the outward rotators produces a permanent rotation of the leg, so that the toes are directed outwards, and, from the altered position of the foot, the propulsion of the body in walking is deficient.

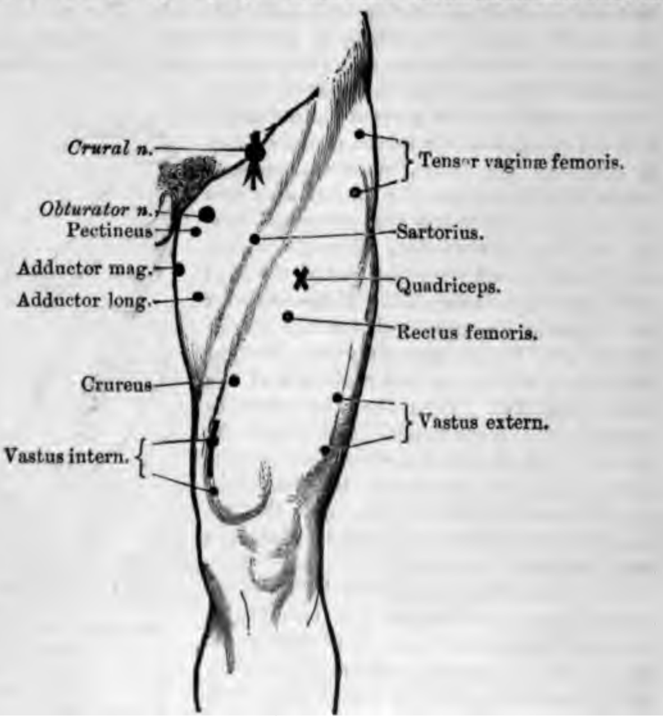

FIG. 25.—Motor points on the front of the thigh (Erb).

The *Pyriformis, Gemelli, Obturator internus*, and *Quadratus femoris* (special nerves from the sacral plexus, but from L. 5) all rotate the thigh outwards, and the first-named muscle, in addition, carries the thigh obliquely backwards and outwards, in the same way as the posterior fibres of the gluteus medius. In paralysis of these muscles external rotation is impossible, and the unopposed tone of the internal rotators (anterior fibres of the gluteus medius and minimus) causes the leg and foot to be habitually turned inwards.

The *Psoas* (special lumbar nerve, L. 2 and 3) and *Iliacus* (anterior crural nerve L. 2 and 3) flex the hip-joint, and, in doing so, cause also a slight rotation outwards. In paralysis, flexion is lost, and the use of the leg in walking becomes impossible.

The *Tensor vaginæ femoris* has a slight power of flexing the hip, and at the same time rotates the thigh in. It normally counteracts the tendency of the ilio-psoas to rotate outwards. If it is paralysed, there is a tendency for the foot to turn out when it is being brought forward in the act of walking.

Adductors of the Thigh (all are innervated from the same roots, L. 2 and 3). —The *Pectineus* (obturator nerve) causes an oblique movement forwards and inwards, *i. e.* a combined flexion and adduction, as in crossing the legs. It also rotates outwards. The *Adductor longus*, and probably the *Adductor brevis* (obturator nerve), have the same action, but the flexion is less than by the pectineus. The *Adductor magnus* (obturator and great sciatic) causes a similar adduction, but while its upper fibres rotate outwards its lower fibres rotate in, and are employed in keeping the foot straight during adduction in riding. This is very difficult if these fibres are paralysed. The foot then turns out when the hip is flexed, either in the recumbent posture or in walking, from the preponderance of rotation out by the other adductors. When all the adductors are paralysed, not only is adduction lost, but in flexion of the hip the foot is moved forwards and outwards, instead of directly forwards, showing that there is normally a synergic action of the abductors and adductors with the flexors in this movement.

MUSCLES MOVING THE KNEE.—*Extensors: Rectus, Vasti,* and *Crureus*, together called the *E. quadriceps* (ant. crural nerve, L. 3 and 4).—The vasti act solely on the knee-joint; the rectus also aids in flexing the hip, but chiefly when the knee is bent. In consequence of its passage over the hip-joint, moreover, the force with which it extends the knee is increased by the simultaneous extension of the hip. This effect is useful in the propulsion forwards of the body in walking. The crureus is unimportant.

In paralysis of the extensors of the knee, standing is still possible if the knee is extended, since the arrangement of the articulation renders a contraction of the extensors unnecessary. But secondary shortening of the flexors is apt to occur, and then standing becomes impossible because the knee cannot be perfectly extended. In the same way, walking is possible if the leg is not moved forward beyond the vertical position; if it is, the knee becomes flexed by the weight of the leg and foot, and the patient falls when he attempts to rest upon it. Rising from the kneeling posture in the ordinary way is impossible. In partial paralysis of the muscles, as in pseudo-hypertrophic paralysis (q. v.), the extension of the knee, in rising, is facilitated by placing the hand upon it, and so bringing the centre of gravity of the body near the fulcrum of the lever formed by the femur. If the vastus internus and rectus are paralysed, the vastus externus may dislocate the patella by the obliquity of its traction. The vastus internus never does so in the opposite condition, because its action is less oblique.

Flexors of the Knee.—These are all supplied from the same spinal roots, although through various nerves—L. 4 and 5, S. 1.—The *Sartorius* (ant. crural nerve) flexes the hip- and knee-joints, and has a feeble power of rotating the thigh outwards and the knee inwards. It is a muscle of small importance.

The *Gracilis* (obturator nerve) adducts the thigh more powerfully than it flexes the knee. It rotates the leg inwards.

The *Semitendinosus, Biceps,* and *Semimembranosus* (great sciatic nerve) are not only flexors of the knee but extensors of the hip-joint, and are the muscles that extend the hip during ordinary walking, the gluteus maximus (q. v.) being called into action only during special efforts. The leg is rotated inwards by the semitendinosus, outwards by the biceps.

In paralysis of the flexors the resulting loss of the power of flexion interferes with walking, since the knee-joint cannot be bent, in the forward movement of the leg, until the thigh is flexed sufficiently to permit the weight of the foot to flex the knee. To prevent the toes striking the ground the foot is unduly flexed on the leg. The loss of the support that the flexor tendons give to the knee-joint leads to an undue strain on the ligaments, which become stretched, and slight retroflexion of the joint may occur.

In paralysis of the muscles that extend the hip, there is a tendency to fall forwards in walking. To counteract this the trunk is carried backwards, and a fatiguing strain on the flexors of the hip results.

In paralysis of the biceps, the leg, during flexion, is rotated inwards; when the biceps remains and the other muscles are paralysed, there is an undue rotation outwards. The effect of these abnormal movements on the ligaments of the joint is such that, after a time, the amount of rotation becomes greater than is possible in health.

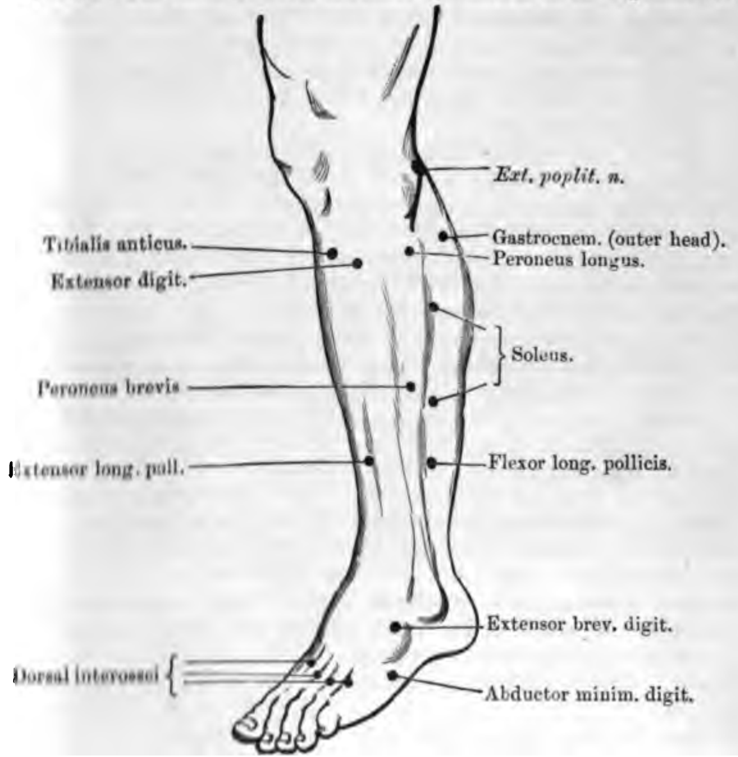

FIG. 26.—Motor points of leg, outer side (Erb).

The *Popliteus* (internal popliteal nerve) has but a feeble power of flexing the knee. Its chief action is to rotate the leg inwards when the knee-joint has been flexed.

MUSCLES MOVING THE FOOT.—*Extensors of Foot on Leg.**—The *Gastrocnemius* and *Soleus* (internal popliteal branch of the sciatic—L. 5 and S. 1) have the same action. They extend the hinder part of the foot and draw down the outer side of the forepart of the foot, but very little the inner side. Hence the foot is rotated, so that the dorsum looks outwards, while the whole foot is turned inwards on the axis of the leg. The peculiar inversion and adduction that thus accompanies extension is due to the form of the articular surfaces. The gastrocnemius has very little power of flexing the knee, but the extension

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* These muscles are sometimes termed "plantar flexors," because they are homologous with the flexors of the wrist. The term is a bad one, since it involves a use-

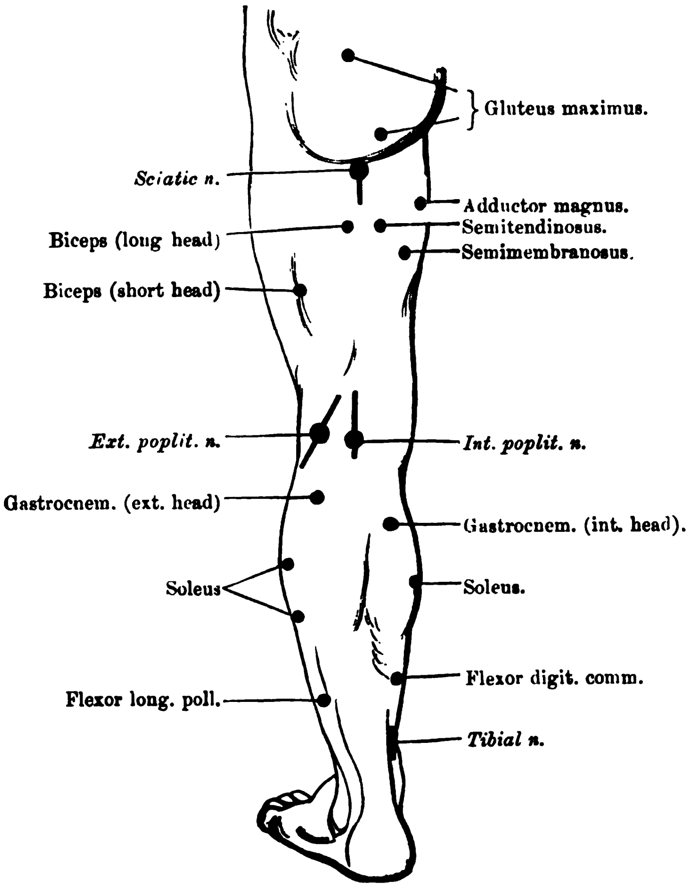

FIG. 27.—Motor points, back of thigh and leg (Erb).

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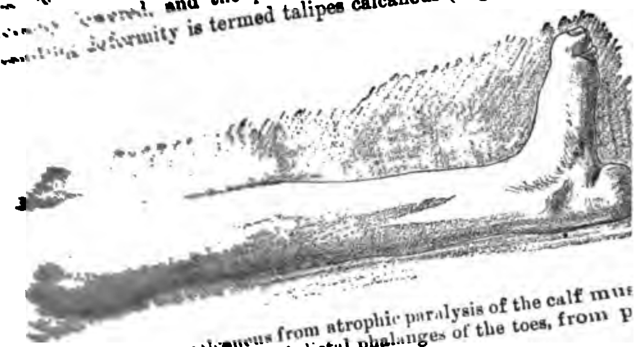
of the word "flexor" in absolute contradiction to its proper signification. We ought not to frame a descriptive term such as this on an analogy which involves a contradiction to the description. Flexion is bending, a movement from a straight line; and extension is less bending, a movement towards a straight (stretched, extended) line. To call a movement toward a straight line "flexion," because in the arm the similar movement produces this effect, is a process that is not description, but the statement of an analogy—the worst possible kind of nomenclature. Names should be descriptive or they should be arbitrary. Neither theory, nor analogy, nor homology should have any part in them. What we call homology is merely a species of analogy; it rests on inference and reasoning, not on simple absolute aspect. Nothing that rests on reasoning is inherently stable and free from change.

the knee increases the effect of the muscle on the ankle-joint, especiall
just as we have seen that the extension of the hip augments the f
the contracting rectus extends the knee. For direct extension o
the peroneus concurs and opposes the inversion. The only diffe
the gastrocnemius and soleus is that the latter, having no attach
can extend the ankle when the knee is flexed as well as whe
paralysis of these muscles, extension of the ankle (b
and flexor longus digitorum) is extremely feeble, and th
carried beyond a right angle. Walking is greatly int
tiptoe is impossible. The unopposed peroneus
of the foot, lowers the head of the first metatarsal bo
plantar arch. In time the ankle-joint becomes over-flexed,
and the plantar muscles and fascia become sh
deformity is termed talipes calcaneus (Fig. 28).



calcaneus from atrophic paralysis of the calf mus
middle and distal phalanges of the toes, from p

(musculo-cutaneous nerve, from extern
everts the foot, lowering the inner border
plantar arch. It also turns the whole
very feeble power of extending the
the foot during extension by the cal
of this muscle the inner part of the
and yields to slight force. T
that the sole is directed inwards, in
sural muscles. The inability to pres
against the ground leads to over-a
plantar arch is lessened; there is "f
(Both are supplied by the anterior
L. 5 and S. 1.) The *Tibialis an*
it elevates the inner part of
flexes the ankle-joint, and
besides extending the toes. fl
consequence of the outward p
These two muscles to
with adduction or abduction.
Paralysis of either weakens fl
flexion being accompa
remains. The defect in flexi
the loss of the instinctive f

is brought forward in walking, causes the foot to catch against the ground. Paralysis of the flexors is followed by secondary contracture of the extensors, and talipes equinus results (Fig. 29), which is the greater the longer the palsy has lasted (Fig. 30). Its occurrence is facilitated, in many cases of palsy, by lessened growth of the bones of the leg, so that the ball of the foot only touches the ground when the foot is extended. There is usually slight rotation inwards of the foot at rest, even when the tibialis is paralysed (see Fig. 29, A), because such rotation is produced by the sural extensors (p. 42); but in this case the slight valgus at rest is changed to varus on an attempt to flex the ankle (Fig. 29, B).

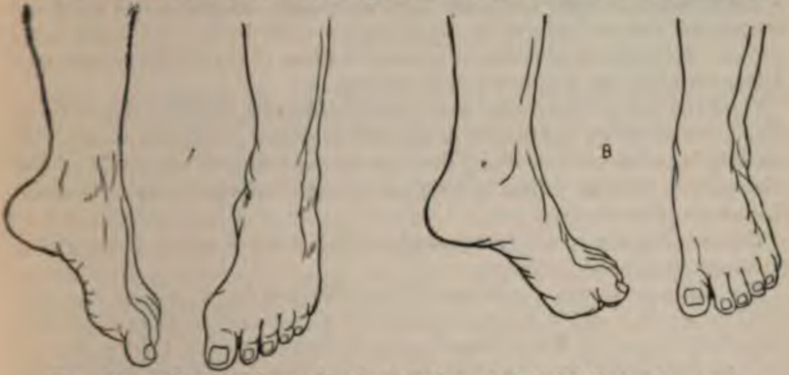


FIG. 29.—Talipes equinus, due to atrophy of the tibialis anticus and secondary contracture of the calf muscles. In A the foot is shown at rest; there is slight equino-varus. In B it is shown during flexion, and the varus is changed to valgus by the action of the peroneus longus. Note the increased extension of the toes in B from the compensatory over-action of the long extensors of the toes. (After Duchenne.)

FIG. 30.

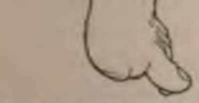


FIG. 31.

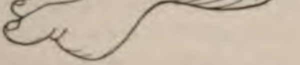


FIG. 30.—Extreme talipes equinus from old-standing palsy of the tibialis anticus (infantile paralysis) and extreme contraction of the calf muscles. No flexor movement was possible.

FIG. 31.—Paralysis of the interossei and the adductor and short flexor of the great toe. The first phalanges are over-extended and the second are flexed, while the hollow of the sole is increased. (After Duchenne.)

The *Peroneus brevis* (musculo-cutaneous [peroneal] branch of ext. popliteal nerve—S. 1 and 2) abducts the foot and rotates it, raising the outer edge.

The *Tibialis posticus* (posterior tibial nerve from int. pop.—L. 5 and S. 1) adducts the foot and curves it, rendering the outer border and instep more convex. Its power of adduction is greater than that of the tibialis anticus, and it does not rotate the foot in the same manner.

These two muscles alone have the power of adducting and abducting without flexing or extending; and in their paralysis these simple movements are lost. If one only is paralysed, a deformity develops corresponding to the action of the other muscle,—talipes valgus in paralysis of the tibialis posticus; t. varus in that of the peroneus brevis.

The *muscles moving the toes* present, in their mode of action, a close correspondence to those of the fingers.

The *Extensor longus digitorum* and the *Extensor longus pollicis* (anterior tibial nerve—L. 5 and S. 1) extend chiefly the first phalanges, while the *Flexor longus digitorum* and *Flexor brevis* (posterior tibial nerve—S. 1 and 2) flex the last two phalanges. The *Lumbricales* and the *Interossei* (post. tibial nerve by ext. and int. plantar—S. 1 and 2), together with the *Abductor* and *Flexor brevis minimi digiti*, oppose both the other extensor and flexor muscles, flexing the first phalanx and extending the others. This action is of great importance in walking, since they give the last propulsion to the body as the ball of the foot leaves the ground. The *Abductor*, *Adductor*, and *Flexor brevis pollicis* (plantar nerves from post. tibial—S. 1 and 2) have a similar action on the great toe, but with adduction or abduction respectively. The interossei also produce a lateral movement of the toes, but this action is of little practical importance. In paralysis of the common extensor of the toes, and of the proper extensor of the great toe, the tonic force of the interossei and analogous muscles produces persistent flexion of the first phalanges and extension of the others. If the conditions are reversed, and the latter muscles are paralysed, the first phalanges are over-extended, sometimes even subluxated, and the two other joints are flexed, so that a claw-like form of foot is the result (Figs. 31 and 28). The final propulsion in walking, above described, is much interfered with, and the attempt is painful because the ends of the toes are turned towards the ground.

Other examples of the effects of paralysis of the muscles of the arm and leg are given in the illustrations to the chapter on Infantile Paralysis (Acute Polio-myelitis).

THE GENERAL CONSTITUTION OF THE NERVOUS SYSTEM.[*]

Our conceptions of the elementary arrangement of the nervous system have been changed during the last nine years by important histological discoveries, which clear many obscurities, give new significance to facts before imperfectly perceived, and involve new pathological conceptions, general and special. Although new difficulties have arisen, as they must do at each step forward, numerous facts, which before were mysterious, become intelligible, and their elucidation constitutes confirmation of the truth of the discoveries. The evidence has been received as adequate by all physiologists. Pathologists must therefore accept them, and reconstruct their conceptions. It is accordingly necessary to give an outline of the constitution of the cerebro-spinal nervous system as at present discerned, even of that which is not of direct present application to pathology, but which may become so and is essential for the perception of the whole. The facts have been found also to be true of the sympathetic system.[†]

These discoveries are the result of a method of metallic staining, first devised in detail by Golgi of Pavia, in which silver is reduced in the structures that have been impregnated with chromium during hardening, and usually also acted on by osmic acid. It displays the minute structure of the grey matter with a distinctness and in a manner that reveal facts before altogether unseen.

Almost the first definite steps were the results obtained in the Invertebrata by Nansen, before he turned his face from polar cells to Polar seas. But the first important disclosures, which involved the radical change in conception, were due to Ramon y Cajal of Barcelona[‡] and to the veteran v. Kölliker,[§] supplemented and diffused by Waldeyer.[‖] Since 1890 there has been incessant work at the subject, and y Cajal has presented to us the chief facts that had then been ascertained, in the Croonian Lecture to the Royal Society in 1895.

All nerve-fibres are prolonged processes of nerve-cells. They consist of an axis, with a "medullary sheath" around it when the course is long, either within or outside the central organs. Most cells have but one such medullated process, larger than the others; some have two. The other processes are short; they soon divide and branch within the grey substance: the long medullated processes also at last end by dividing and ramifying.

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[*] The interpolation of this account of the recent revolution in fundamental elements of our knowledge, although not quite consistent with the plan of the book, seems the most convenient way of presenting the facts to the reader. They pass below, and rise above, the range of practical knowledge which can be used in common work, and yet are changing our physiological and pathological conceptions in a manner and degree which must be adequately recognised, although much of their effect is still uncertain.

[†] v. Kölliker, "Histol. Mittheil.," 'Wurzburg. Sitzungsb.,' Nov. 23rd, 1889.

[‡] Previously, 'Internat. Monatschr. f. Anat.,' 1890, Bd. vii.

[§] Loc. cit.

[‖] 'Berlin. med. Wochenschr.,' 1891, No. 28.

The conception formerly held may be thus stated. Of the ultimate divisions of the short, quickly-branching processes in the grey matter, some had been thought to join the terminations of similar processes from other cells, either neighbouring cells in the grey matter, or the terminal branches of medullated nerve-fibres, processes of cells far away. Thus, of the motor cells of the spinal cord, some of the processes, passing backwards, were believed to join those of sensory cells in the posterior cornua, of which the medullated process was a fibre of the posterior root. The constituent elements of the nervous system thus formed, by branch union, one continuous complex network, with paths for the nerve-impulses, due to union and continuity of the cell-processes. The paths in actual use were determined not only by such union, but also by differences in "resistance" among those which continuity provided. The "resistance," which thus permitted an energetic impulse to spread more widely than a slighter one, was varied in degree by functional activity; it was diminished by the repetition of the same activity, and it was also varied mysteriously by other nerve-impulses from various sources It might thus be increased so as to "inhibit" action. In so far as any attempt was made to conceive its seat, it was thought of as in the cells, or in the feltwork of uniting processes in the grey substance, called "spongy," from its blending trabeculæ.

Much of this conception is retained. But the more the methods of examination were improved actual union of the branches of cell-processes became less perceptible under the microscope. The development of the use of staining agents, especially metallic, has increased the power of discrimination. Variety of aspect can be thus produced in structural elements that before were indistinguishable. By the use of such methods, and especially that of Golgi, the branches of the processes and fibres have been clearly traced, and they have been found not to unite. They are distinctly seen to end in the structural material in which the nerve-cells lie, sometimes by an enlarged knob-like extremity, sometimes by a point. Often the branches cross and even interlace, with the semblance of union, but close observation shows that it is a semblance only. Discontinuity is found to be the general rule. Whether invariable or not is still undecided; it is most difficult to exclude union in the dense felt of fibrils which the branching processes form in some parts, the "neuro-pilema" of His, and, moreover, the general rule of free endings is compatible with occasional actual union.*

From this it follows that the "nervous system" consists of discontinuous elements, each a cell-body with its processes, long and short. For these elements the name "neuron," proposed by Waldeyer,[*] has been all but universally adopted; for distinctiveness, its plural is formed according to the living language, and not the classical form—in English it is "neurons," in German, "neuronen," in French, "neurones." The chief process, the medullated, or axis-cylinder process, is called the "axon," or "axis-process."[†] The branching processes in the grey matter are termed the "dendrons," the branches of these "dendrites."[‡]

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* Cf. Masius, 'Arch. de Biol.' 1892; Fritsch, 'Brit. Assoc. Report,' 1892.

From the axons there often spring fine fibres, which pass off at right angles, termed "collaterals" by Ramon y Cajal, the relations of which are imperfectly known. They have been seen to end by a T-like division and ultimate branching.

The definite and extended discernment of another fact deepens the importance and application of that just described.

Thirty years ago[§] Max Schultze discovered and depicted the fact that the axis-cylinder is compound and not simple, that it consists of a large number of fibrils,—"primitive fibrils," he termed them. He pointed out that they could be traced throughout the fibre, separated by a finely granular substance. They could be well seen where the axis-cylinder widens in joining the nerve-cell, that is, where the cell narrows into its chief process. Others could not see the significant striation, among them Ranvier, whose work dominated science throughout the next fifteen years,[||] and the fact was practically ignored. The axis-cylinder was regarded as simple and integral, until recent methods have enabled the clear establishment of the correctness of Max Schultze's observations. The number of fibrils that constitute an axis-cylinder is considerable. Obersteiner has found that there are about fifty in an axis-cylinder of the sciatic nerve of the frog.[¶]

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[*] Loc. cit.

[†] Because the "axon" is the separate "nerve," when one exists, an attempt has been made to make current the use of "neuron" for this alone. But etymological consistency has little influence on the vitality of names. The use of "neuron" for the whole element has become so general that resistance to it is futile. Moreover, the conception attached to it in use is already definitely detached from its etymology. Lastly, although the cell-body and its processes are one, to have only the word "cell" for the whole element, a word that will still, inevitably, be applied to the cell-body, leaves the latter without nominal distinction from the other two parts of the element—the neuron and the dendrons. Hence the word "neuron" is here used in the established senses.

[‡] "Dendrite" has also been used as equivalent to "dendron," and still is; but the need for distinguishing the twigs from the process itself makes it highly probable that this convenient separation of the words will become universal.

[§] In 1868; see Stricker's 'Histology' (New Syd. Soc. trans.). The observations were confirmed by Babuchin in 1868 and 1869.

[||] See further on Structure of the Nerves, p. 62.

[¶] Personal communication.

These fibrils consist of a [illegible] substance, the "hyaloplasm" of Leydig and Nansen, so termed from its greater translucency in hardened tissue. This is isolated by a slightly granular material, distinguishable chiefly by its difference of aspect and staining after the changes produced by hardening agents. It has been termed the "spongioplasm"—"plasm" because it also has a soft consistence, "spongio" because it is said to form trabeculæ, which constitute the chief element in the ground substance or matrix of the "spongy" grey substance* in which the cells lie and their processes branch.

From this compound constitution of the axon we must infer that its terminal division into branches is merely the separation of these fibrils, first into groups and at last into single fibrils—"primitive fibrils." It is so at the periphery and in the grey matter. The final twigs may be termed "axites," if a special designation seems needed.+

The short branching processes have been found to consist of similar fibrils, and this is true also of those supposed to be extensions of the substance of the cell and therefore called "protoplasmic processes." This name should be given up with the conception attached to it. The opinion that their function is to convey nutritive material to the cell-body was, indeed, never more than an hypothesis, resting on no real foundation.‡

With the recognition of the fibrillary constitution of the axon and dendrons, has been associated an equally important recognition of the fact (most clearly depicted by Max Schultze §) that their fibrils pass through the body of the nerve-cell without interruption. Those of the axon diverge to pass to the several dendrons, but those of the latter do not pass all to the axon. Some pass directly to other dendrons, so that, in such, there must be conduction from the cell as well as towards it. Moreover, both y Cajal and Kölliker have observed, in certain cells of the cerebellum, all the fibrils of an axis-cylinder pass directly to a dendron, scarcely entering the body of the cell.

Twigs from such processes of small cells, especially in the Invertebrata, have been said to join the elements of the neuroglia.¶ The fact is in harmony with the origin of both from the same embryonal tissue. The differentiation of the nerve-elements from this, may leave some blending of structure. It must be remembered that there is much uncertainty regarding the nature of many of the smaller cells, and it is conceivable that some are nervous and some neuroglial, and that there may not be always actual separation of their connections.

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* Apathy, 'Biol. Centralbl.,' 1889. The term "spongy grey substance" was before in use on account of the sponge-like interlacement of the cell-processes. The conception attached to it has thus become somewhat confused.

+ Especially since one writer has termed them neurites, which would be confusing unless the axis-process were termed the "neuron," as it certainly will not be. The only proper use of this word (neurites or neuronites) would be for the terminal twigs in general.

‡ That these dividing branches, long, and not obviously permeable, should be the channels by which the cell receives that which could pass directly through its walls, was simply a positive inference from the negative fact that no other function was obvious. That we must cease to consider these processes "protoplasmic" and nutritional was insisted on in 1890 by Rabl Ruckhardt ('Neurolog. Centralb.').

§ Loc. cit.

‖ v. Kolliker, 'Wurtzburg. Sitzungsb.,' Nov. 23rd, 1889.

¶ Among others by Ladowsky, 'Verhandl. Med. Congress,' Berlin, 1890, ii, 92.

An essential difference between the axon and the dendrons cannot as yet be established. Branching does not differentiate them, for the axon also branches after a longer course. Nor does either multiplicity or length. The cells of the posterior ganglia of the cord have but one axon and one dendron, united for a short distance as the single process of the "unipolar" cell. These two processes are of nearly equal length in the case of those cells that give rise to the fibres of the posterior median column; these, reaching the cord by the posterior roots, ascend to the level of the medulla, while the other division of this cell-process probably comes from a muscle as far away. We see also, in this instance, the absence of any real distinction from the direction of conduction. Although the axons of the motor cells conduct from these, so also must some of those dendrons to which fibrils pass from others. Moreover, in the case of the posterior root-fibres which go to the local grey matter, the long fibre from the periphery conducts towards the cell; the shorter fibre, which soon branches in grey substance, conducts from it. Opinions may differ as to which should be regarded as axon and which as dendron, but analogy suggests that the latter should be that which soon ends in the grey substance. But it is difficult to avoid regarding each fibre of the cells related to the posterior median column as an axon. There is no reason for regarding either a dendron or an axon as an essential element of a cell. Yet the distinction is necessary, and the general application of the names is justified by the common difference between the one medullated process, which passes out of the grey matter, and the other numerous processes, which are not medullated, and soon divide within the grey substance.

The extent to which the physiology of the nervous system is inferred from its minute anatomy, is illustrated by the revolution in our conceptions produced by the discovery of the discontinuity of its elements, the fibrillary constitution of the conducting structures, and the course of the fibrils. These, passing through the nerve-cells, without interruption, can merely conduct through the cell-body as they do elsewhere in their course. With this fact disappears the old idea, so simple, apparently so adequate and reasonable from its superficial analogies—the idea that the nerve-cells are the seat of the production of nerve-impulses, that in them "nerve force" is generated from the latent energy stored in the nutritional compounds which have entered into their constitution. But we cannot have such generation where the fibrils are continuous. For it, we must look to their extremities, formerly supposed to be in the cell-body, now perceived to be in the "spongy grey substance." The process must be where the continuous fibrils begin in the centre, as it is in the skin and other parts of the periphery, in the case of fibrils that conduct thence. As all the nerve-impulses that reach the centres, through which external influences act, and the environment is perceived, originate in the minutely separate nerve material at the extremity of the afferent fibrils, so all the outgoing impulses, and those which pass from one part of the centres to another, originate in their special form at the analogous extremities of the fibrils in the grey substance. We must conceive these impulses arising in the extremities of the "cellulipetal" dendrites (to use the somewhat cumbersome term of v. Kölliker), being excited in some way by the stimulus of the impulses which reach the contiguous extremities of other "cellulifugal" fibrils, dendrites or branches of the axon of a distant cell. The incoming impulses may excite those that go out, as impulses are excited at the periphery by other forms of energy; or there may be a process of conduction, through the intervening ground-substance,—the matrix in which the cells lie and the dendrites end. For the increase in the amount of nerve-energy which often takes place, there must be more than conduction, its production must be "excited" by that which is conducted. We can conceive such excitation without continuity, especially when we consider that the stimulation of muscular protoplasm is by the impulses in nerves which terminate on the fibres, and not in continuity with the contractile substance—indeed, separated from it by a structure apparently quite different in its simpler nature.

If this conception of the origin of the impulses in the terminations of the fibrils seems less easy than their origin in the more massive nerve-cells, we must remember how multiplicity neutralises minuteness. Indeed, the minuteness of the conducting fibrils almost involves a multiplicity of minute sources of nerve-impulses,—which must, moreover, be far from minute in comparison with the molecules, from the latent energy of which the impulses proceed. All modern discoveries prepare us for finding every mass to be an aggregation, and each perceptible quantity of energy to be made up of minute constituents, every one of which is definite, both in its separate character and in its contribution to the effect of the whole. The finely divided nerve-substance will constitute a total source of energy, as adequate in extent as a nerve-cell, with a more obvious facility for the renewal of the molecules lost in functional action, and far greater opportunity for varying relation to the structures from which the impulses are received.

What, then, is the function of the nerve-cell? The one certain fact, established by evidence that is secure, is that on it depends the life of the nerve-fibres—the vitality of all the processes, equally that of the dendrons and of the axon. The evidence of this, the immediate degeneration of any separated part, is well known. It is less conspicuous in the case of the dendrons, but sufficiently certain. How the influence is exerted we can only surmise. The nucleus of every cell is its life centre, and in some way the nutrition of the protoplasm is determined by it. The cell-body has a complex structure, dimly perceptible by the help of reagents, at present beyond our practical comprehension. One distinct fact, however, is that the protoplasm of the cell, extending into the narrowing axon, thins away between the outer sheath and the white substance, becoming unrecognisable until one of the many nuclei is reached. Each of these is surrounded by like protoplasm, also thinning off along the fibre until invisible. It is certain also that if the cell-body and nucleus are destroyed, or the fibre separated, the process of degeneration begins by changes in these nuclei. They seem to be concerned in carrying on the influence of the distant cell, and it is not easy to conceive any other path for this influence than a thin layer of protoplasm, within the sheath, continuous from nucleus to nucleus, vitally influenced by these, and conveying, by its own nutritional state, that of the cell. A space, in this position, is rendered visible by reagents which make the contents of the sheath shrink. Indeed, coagulated material, in small quantity, can be seen within it.* This has been regarded as evidence of the presence during life of such a space as is here assumed to contain connecting protoplasm.

Many facts, previously obscure, become more intelligible in the light of the new discoveries. The knowledge that the cell governs the nutrition of the fibre, and the belief that from it proceeded the nerve-impulses, involved a correspondence in the direction of nutritional influence and of conduction, and it was found, in fact, that conduction and degeneration were in the same direction as a rule—but only "as a rule." It was untrue of the sensory nerves. These, coming from the cells of the ganglia on the posterior spinal roots, degenerate downwards, but they conduct upwards—an anomaly which had to be simply ignored. The fact is now seen to be in harmony with others.

The arrest of secondary degeneration in the grey matter, the fact that the degeneration of the fibre which enters the grey matter never passes on to the next nerve-cell, through which conducted impulses must pass, was mysterious when they were believed to be in continuity. The discontinuity explains it at once. The degeneration is limited to the single neuron.

The division of the axis-cylinder at its terminal ramification, whether that of the sensory nerve in the skin or of the motor nerve in the muscle, was scarcely intelligible when the axis-cylinder was regarded as a single conducting path; it is so when we perceive that the apparent division is simply the separation of its constituent fibrils, each a distinct conducting path. This fact is, moreover, especially important in relation to the minute localisation of the various forms of sensation in

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* Schiefferdecker, 'Arch. f. mik. Anat.,' xxx, 1887.

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impossible, and it is not clear that actual conduction of nerve-impulses is incompatible with the augmentation we include under the term "excitation." An increase must occur when, for instance, in many reflex actions the afferent impulse that reaches the spinal cord is minute in proportion to that which leaves the centre. We seem thus to have a process of stimulation comparable to that in the cutaneous nerves (themselves apparently analogous to dendrites), under the influence of some other form of energy from the outside. In this connection it is especially noteworthy that many dendrites terminate in knob-like enlargements, suggesting receptive functions.

We do not know the nature of a "nerve-impulse." We must, however, assume that it is energy in some form of motion, released from that previously latent in the nerve-substance, apparently the "kinetoplasm" or "hyaloplasm." Chemical change occurs by which simple compounds are formed, as in all similar processes, and the simplest conception is that the process is like that in a train of explosive material, modified, restricted, but perhaps accelerated by the influence of life.*

The motion of a touch, which acts upon the nerve-endings in the skin, reaches them by passing through the skin as the simplest form of motion; so also pass the special forms of wave motion that constitute electricity and heat. A differentiation of the nerve-tissue in the ending seems to enable their molecules to receive one special form of motion most readily. Hence our "special senses." The manner in which the equilibrium of the nerve-substance is disturbed in each doubtless differs much, but it is instructive to note in connection with the theory of the nerve-impulse just suggested, that the wave-motion of light seems first to induce recognisable chemical changes in material of unknown nature.

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* This view has been developed at length in the writer's 'Dynamics of Life,' London, Churchill. 1894, but was clearly stated by Herbert Spencer, 'Elem. Biology,' vol. i, 1864.

clear cementing substance, shown by the fine dotting in the figure. There is one nucleus (n) to each internode, about its middle; hence they are sometimes called "internodal nuclei." Around the nucleus is a little protoplasm, and it is probable that a very thin layer of protoplasm everywhere lies between the sheath and the myelin connecting that of the adjacent nuclei, and these with the protoplasm of the nerve-cells (see p. 59). The internodes are shorter towards the termination of a nerve. It is important not to confound these divisions with other imperfect indentations (IS, Fig. 32, I), often called the "incisions of Schmidt," or "indentations of Lautermann."* These are oblique, incomplete divisions of the white substance. Many exist in each node. They are believed by some histologists to be of artificial origin, but their uniformity is in favour of their dependence on some structural feature.

The myelin, Ranvier suggests, must protect the axis-cylinder, since its almost liquid consistence will diffuse pressure on the nerve. Other possible uses it subserves may be suggested, but we have no proof of them. The nodal segmentation evidently permits nutrient material to reach the axis-cylinder.

The "grey fibres," or "non-medullated fibres," consist of an axis-cylinder, sheath, and nuclei (Fig. 32, III), but contain no myelin. They constitute the sympathetic nerves, but some (also probably sympathetic fibres) are found in all the spinal nerves. They are absent from the nerves of special sense except the olfactory, which contains no other fibres.

The nerve-fibres are united into "fasciculi" by delicate nucleated connective tissue, and these fasciculi are similarly connected into larger bundles, while the whole nerve is surrounded by a dense connective-tissue sheath or "perineurium." Under this sheath, and extending between its lamellæ and amongst the individual nerve-fibres, there is a lymph-space lined by flattened endothelial cells, which probably plays an important part in the propagation of inflammation. From the perineurium a very delicate "sheath of Henle" extends on to the ultimate divisions of the nerve. All these tracts of connective tissue and spaces are continuous, and they convey the blood-vessels. They also contain nerve-fibres, "nervi nervorum," which are derived from the nerve itself.† The sheath and connective tissue of each nerve are thus part of the area of distribution of its own fibres.

PHYSIOLOGY OF NERVES.—The first great fact to be kept in view is that the axis of each nerve-fibre is the prolonged process of a nerve-cell, the wall of the cell and process being continued as the sheath of the nerve-fibre, the protoplasm of the cell becoming less and less as the process narrows, and the white substance, the medullary sheath, making

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* They were first described by Schmidt of New Orleans, and afterwards by Lautermann.

† Horsley, Roy. Med. and Chir. Soc., January 22nd, 1885.

has reduced the process almost to
medullary sheath begins in the layer of
the axis within the neurilemma, and which
the myelin increases, for some distance.
cease on either side of the myelin
medullated fibres there is a continuity
neurilemma, and probably the protoplasm,
from the fact that the fibre is by
cell; while it is moreover found to be so
It shares all modifications in the nutrition
a perfectly normal state if the nutrition
Yet the relation is less simple than it seemed
to be but an attenuated prolongation of
It has been pointed out in the last chapter
the axis pass through the cell and,
considered part of the cell, we are led to look
prolonged along the fibre, for the element of
in harmony with much elsewhere.

fibres is seen in a different aspect under the
As already explained, it was formerly
and the fibres conduct what is termed
fibres also can generate this when they are
conduct the impulse that passes through them.*
that the nerve-fibrils simply conduct when
cell body, as they do elsewhere; and it is con
a bundle of fibrils simply skirts the edge o
in passing from one process to another, a

fibril must be true of all. The relatio
conception of differences. If any fibr
substance, all would do so. If it is true
all, that the function of the cell is vi
begin as such, as conducting structu
susceptible to the same changes through
is a change of constitution or of struc
the source of the energy that is condu
how to look.

where they terminate, as in musc
be regarded as separation or union of

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function of the cells and the fibres was
this book in words that are worth quoting,
facts of function as well as of structure
must conduct, and the cell must itself
of the path through which the ner
passes through must be conducted.
if it is renewed, we cannot so distin
fibre as to say that there is not cond

them is greater than normal, and is granular; there is in places a local increase in the amount of protoplasm within the sheath, compressing the myelin (A *x*, B *x*, C *x*). The nuclei then become detached from the sheath; the protoplasm everywhere increases, and encroaches on the myelin, until here and there it meets across the tube, com-

FIG. 33.

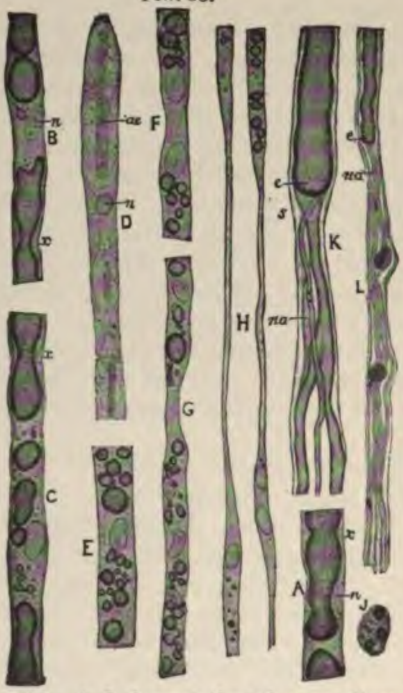

FIG. 34.

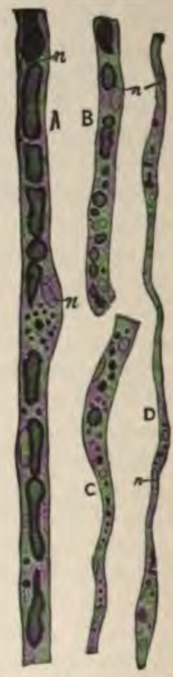

FIG. 33.—A–J, degeneration of nerve-fibres (osmic acid and carmine staining). A, from sciatic of rabbit four days after section; B, C, same, fifty hours after section; D, a fibre stained with carmine only, to show the axis-cylinder; E, F, G, fibres from pigeon three days after section; H, two fibres from pneumogastric of rabbit six days after section; J, a lymphatic cell from interstitial connective tissue, containing globules of myelin which it has taken up. In all the figures—*n*, *n*, nuclei; *x*, *x*, constrictions of the myelin produced by the growth of the protoplasm; *ac*, axis-cylinder.

K, L, regeneration of nerve-fibres. K, from pneumogastric of rabbit seventy-two days after section; L, from sciatic of rabbit ninety days after section; *e*, rounded end of white substance of central end of nerve; *s*, sheath; *na*, new axis-cylinder. In L are two globules of myelin remaining from the degeneration of the old fibre.

FIG. 34.—Degenerating fibres from cutaneous nerves of man. (After Pitres and Vaillard.) A, from near a bedsore in a case of fracture of the skull; B, C, D, from the fifth nerve in a case of neuralgia and ulceration of the lip; *n*, *n*, nuclei. In A the protoplasm and nuclei are increased. and the myelin is breaking up, the processes having proceeded furthest in the neighbourhood of the nucleus in the middle of the fibre; in B the segmentation has gone on to the formation of globules, which in C are, for the most part, small, and many have been removed, so that the fibre is narrow; while in D all the products of degeneration have been removed from considerable tracts of the sheath.

pletely separating the myelin (A, lower part; B, upper part), and with the myelin the axis-cylinder (D). This process then goes on with increased rapidity; the myelin is broken up into smaller and smaller fragments (C, E, F), which become globular in the watery protoplasm, just as oil forms globules in water. The nuclei meanwhile continue to enlarge, and then divide, first the nucleolus and then the whole nucleus (F). The two nuclei may again divide, until (as in G) there are four or more nuclei in each internode, instead of one only as in health. The small globules of fatty myelin seem to become changed in chemical composition, since they are stained less deeply by osmic acid. Ranvier suggests that their fatty matter may undergo a process of saponification. Ultimately they seem to pass through the sheath, are taken up by connective-tissue cells and lymphatic cells in the vicinity (as in J), and are gradually, for the most part, removed. By the time the myelin is in small globules the nuclei cease to multiply. On the removal of the products of degeneration the sheath shrinks, and looks empty in places; but here and there it is enlarged by the nuclei, protoplasm, and a few remaining myelin globules (H). Hence in transverse section many small sheaths are seen, with a few of larger size where they have been cut across at these swellings.

In the rabbit the first changes are visible at the end of twenty-four hours; the first complete interruption of the myelin and axis-cylinder occurs about the end of the second day; the process of destruction is considerably advanced at the end of the fourth day, and is finished, and the multiplication of the nuclei ceases, towards the end of the second week. In peripheral nerves the changes seem to progress from the lesion to the periphery, but within the central nervous system they are said to begin at the same time in all parts of the fibre. It is uncertain to what extent this difference is real or is only apparent, but the process goes on most rapidly at the periphery.

At the end of the second day, in the rabbit, stimulation of the nerve by electricity below the lesion no longer causes muscular contraction, the disappearance of electrical excitability coinciding with the first complete segmentation of the myelin and axis-cylinder.

A curious fact, unfortunately more interesting in pathology than in practice, is that the degeneration of a divided nerve does not occur if the ends are brought quickly into perfect apposition. It is so in animals, and probably also in man.* Immediate replacement may occur in some incised wounds, and thus the fact is explained that simple division is a less serious injury than a grave contusion.

The axis-cylinder seems thus to undergo these changes as a whole, although we know that it is a group of fibrils. But we have seen that the conducting substance of these fibrils, and that which separates it (and may be conceived as analogous to the myelin around the whole axis-cylinder), differ but slightly in consistence. Both are nearly fluid, and [illegible] break up into globules. Although they differ in constitution in a manner and degree that subserve entire difference in function, and permit reagents to produce [illegible] differences in aspect and character [illegible] if simply transferred to the living state, and yet the [illegible] of an essential difference of absolute importance.

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* See Bowlby, 'Injuries and Diseases of Nerves,' p. 32.

The process [illegible] is the result of an active growth of the nuclei and protoplasm of the nerve, [illegible] of the cellular elements of which the nerve is composed. Why does this occur? The determining cause is the interruption of the continuity with the cell-body or the destruction [illegible]. Ranvier connects the destructive growth of the protoplasm with the loss of function in the axis, and suggests that normally the [illegible] of this restrains the vital energy of the cell-elements.

These changes are often attended with others outside the fibres, [illegible] as evidence of inflammation. Several observers have described increase of nuclei and accumulation of leucocytes in the interstitial connective tissue, and even in the nerve-sheath, with dilatation of the [illegible]. Such changes are, indeed, intense at the primary lesion; their degree in the nerve below the lesion varies and seems to be proportioned to that in the primary process. This is another [illegible] the important fact that *the irritative character of the* [illegible] *the irritative nature of the primary* [illegible]. We may say, if we like, that in the slighter degrees it is [illegible] in aspect in proportion to the signs of inflammation [illegible] primary lesion. But this is only another mode of stating [illegible] the importance of the transmission [illegible] of the process cannot be too strongly insisted on.

The [illegible] degeneration occurs more slowly in the [illegible] still slower in man, in whom it is [illegible] segmentation does not occur until between the [illegible]. It is certain than an identical process occurs [illegible] peripheral nerves near bedsores and in the [illegible] Pitres and Vaillard, are shown in Fig. 34, a comparison [illegible] Fig. 33 will show the identity of the process. [illegible] after complete division of a nerve in man the [illegible]. But the most common lesion is, or involves [illegible] and the process of degeneration is thus [illegible] of the primary lesion, and in time as [illegible] latter cases, which are chiefly those of focal [illegible] be the same as that which follows section of a nerve. The [illegible] in irritability, which will be more fully described [illegible] of the symptoms, is a slow depression, sometimes moderate in degree. The depression may be preceded by an increase in irritability. In such cases there can be no complete segmentation of the nerve-fibres. There must be a gradual alteration in the molecular nutrition of the axis-cylinder, changing its excitability. Even in severe cases there is not usually a sudden loss of irritability; the current necessary for stimulation has to be made stronger, until at last in seven or eight days the strongest endurable current fails to cause muscular contraction. But we cannot infer, from this alone, that there is an actual interruption of the axis-cylinder. A stronger current might still excite the nerve, because, as we shall presently see, when a nerve is being regenerated, an axis-cylinder may conduct, and still not be excitable by currents of ordinary strength.

All severe changes in the nutrition of the fibres involve the intramuscular nerve-endings in the same degree.[*] The evidence of this is that the faradic irritability of the muscles (which depends on the nerves within them) presents changes quite similar to those of the nerve-trunk. But this is not always true in slight changes of nutrition of the nerve. We shall presently see that the slight alterations of irritability in the nerve and muscle do not always correspond. The nerves terminate in structures of special nature, and these may well have some slight degree of nutritional independence.

These secondary destructive changes occur in the peripheral part of the divided nerve. But the part that remains continuous with the cell does not always preserve a normal state. Changes occur in it, especially near the lesion, which are still the subject of study, and of which more must be known before their significance can be determined. In a divided nerve the changes, which are mainly those of ordinary, often incomplete, degeneration in isolated axis-cylinders, affect the centripetal (sensory) fibres far more extensively than the centrifugal. Later on the cells connected with both suffer, the nucleus shrinking and the protoplasm to some extent becoming disintegrated.[†]

Regeneration may occur in the nerve after the degeneration is over. It is a slow process, occupying the second, third, and fourth month after division. According to Ranvier, and as Waller thought, it occurs always by the growth of new axis-cylinders from the central end of the nerve (see Fig. 33, K and L), which ultimately become covered with myelin. One or more new fibres may spring from each central fibre, and these may subdivide. All are enclosed in a sheath which is continuous with that of the central end (Fig. 33, K, *s.*). We must assume that only some of these axis-cylinders persist and achieve functional permanence. But the whole of this subject needs re-investigation in the light of the multiple constitution of the axis-cylinder.[‡] Sometimes these fibres twist about, and even turn back and grow upwards, probably in the direction of least resistance. In animals new fibres may grow through a considerable extent of cicatricial tissue between the divided ends of a nerve, but in man it is doubtful whether regeneration of a divided nerve occurs unless the extremities are brought in contact, or at least close proximity. Some investigators believe that there is a formation of fibres in the peripheral extremity independently of the growth of new fibres from the central end.* In cases of slight injury, regeneration occurs more readily; in these it is probable, as we have just seen, that degeneration has been incomplete. The regenerated nerve-fibres regain some conducting power while they are still much narrower than normal, and before they can be excited by electricity.†

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[*] Fully described and figured by Babes and Marinesco, 'Babes' Atlas Path. Histol. des Nervensystems,' part i.

[†] See Homen, 'Ziegler's Beiträge,' viii. and 'Babes' Atlas,' part 2. R. A. Fleming ('Brit. Med. Journ.,' 1896, ii, p. 918) lays great stress on the degeneration of the fine (vaso-motor?) fibres above the lesion, and describes also a considerable connective-tissue overgrowth in the same part.

[‡] The precise renewal of these fibrillary conducting paths cannot be ascertained in animals, and in man the amount of damage that is renewed is far smaller. Facts are needed regarding minute differential localisation after recovery from an injury, compared with the normal.

Muscles.—The degeneration of the motor nerves is attended by changes in the nutrition of the muscles. These commence in or after the second week. The muscular fibres become narrower, and may be reduced, ultimately, to one third of their former width. An increase in width has been said to precede the narrowing for a few days.‡ The transverse striation becomes less distinct, and the striæ seem to be nearer together than in health. The fibres may become cloudy or granular, but do not present actual fatty degeneration except in some very acute cases. If no regeneration of the nerve takes place, the transverse striation gradually disappears, and may be replaced by a longitudinal striation, or the fibres may undergo certain chemical changes, and present a peculiar glassy appearance, which has been called "vitreous generation." During the progress of the changes in the fibres, the nuclei of the sarcolemma and of the interstitial tissue are increased in number, and develop into fibrous tissue, so that ultimately the muscular fibres are separated by considerable tracts of connective tissue, and a state of cirrhosis results. If regeneration of the nerve occurs the muscular changes are arrested, and the normal condition is slowly restored. When recovery of the nerve is slow, and the secondary process in the muscle considerable, the fibres remain small, the amount of connective tissue is permanently increased, but undergoes contraction, so that the muscle is for a long time smaller than normal, and its natural bulk may never be regained. If no regeneration of the nerve occurs, the muscular fibres gradually disappear; fibrous tissue takes their place, and, slowly contracting, permanent shortening may result. Similar shortening sometimes occurs when there is partial recovery of the nerve and muscle. In most lesions of nerves, other than actual division, some fibres recover, even though others are permanently destroyed. The muscular degeneration is the result of that of the motor nerves, as described at pp. 66, 67.

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* *E. g.* Neumann, Mayer, &c. A full abstract of their observations is given by Bowlby (loc. cit.), and also by Allen Starr in the Middleton Goldsmith Lectures for 1887 ('New York Med. Record,' February, 1887). By some the new axis-cylinders are said to be formed by elongation of the fragments of the old axis; by others from the nuclei of the sheath of Schwann, but these have been found by others to take no part in regeneration (von Notthaft, see below). But Ranvier's careful investigations have been fully confirmed by the minute researches of Vanlair ('Arch. de Biologie,' 1885), and by the more recent experiments of Stroebe ('Ziegler's Beiträge,' 1893), and it is difficult to understand that perfect axis-cylinders should be formed and remain unexcitable, as the peripheral segment certainly does. Structures may be formed resembling axis-cylinders that are not really capable of the proper function. The weight of Ranvier's undisproved and confirmed observations is very great. His conclusion that there is no discontinuous formation of fibres has been also confirmed by von Notthaft ('Zeitsch. f. Zool.,' 1892, Bd. lv). It is possible that the process, in the peripheral part of a nerve, is influenced by the connection with other nerves and the recurrent influence of anastomosing fibres.

† Ziegler, in a recent paper ('Arch. f. klin. Chirurg.,' 1896), reasserts the abandoned view that the old axis-cylinder plays no part in regeneration, the active agent in which is a nucleated protoplasm which he states to be an outgrowth of the sheath of Schwann (this probably refers to the protoplasm mentioned on p. 59). From this he describes the development of a primary protoplasmic fibre, from which are differentiated not only a new axis-cylinder but also a new sheath of Schwann and medullary substance. This new axis-cylinder is ultimately joined to the old stump. These statements have not as yet received confirmation by other observers.

‡ Steinert, 'Verh. Phys. Ges. Wurzburg,' 1888, No. 10.

Symptoms of Nerve Injury and Degeneration.—The symptoms that attend the lesions of motor nerves and the consequent degeneration are of great importance. The lesion of the nerve causes paralysis of the muscles supplied by it, due to, and in proportion to, the interference with the conducting power of the nerve. The muscles at once become flabby from loss of tone, and to this atony actual wasting is added in the course of a few weeks. The wasting is due to the reduction in size of the muscular fibres. If the sensory nerve-fibres are not interrupted, the muscles become tender to the touch, and pain is caused by their strong contraction, due probably to the effect of interstitial inflammation on the sensory nerves, which end in the connective tissue.

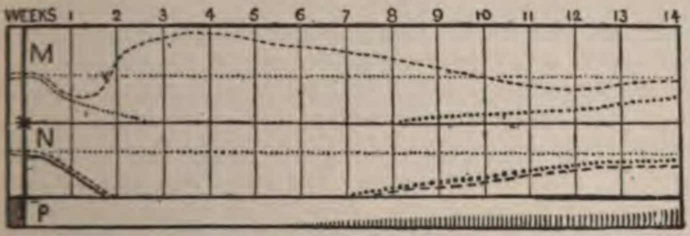

Fig. 35.—Type of degenerative reaction in a case of nerve-injury of moderate severity. (After Erb.) Muscle irritability lowered from middle of first week; faradic, extinct in middle of third, reappearing in ninth; voltaic, increased from middle of second week until tenth, then depressed until fourteenth. Nerve irritability (changed alike to both currents) is lowered from middle of first week, lost at end of second, reappearing at seventh. Power lost until end of fifth week.

The most important symptoms are those that are afforded by electric[illegible]amination of the muscles and nerves, since they enable the degenerative changes to be ascertained and followed during life.* The alterations in the electrical reaction, consequent on this degeneration, have been already briefly mentioned (p. 29), but must now be described in greater detail. The rapid degeneration of a nerve, which follows a severe lesion, is attended by a loss of irritability on electrical stimulation, the loss being the same to faradism and voltaism. After such lesions as are common in man—neuritis, for instance,—there is no sudden loss, such as occurs after injury of a nerve in an animal, when the nerve becomes segmented, but there is a more or less rapid diminution of excitability, and this goes on until no stimulation can be produced, even by a strong current. The progressive changes in irritability may be conveniently represented on a chart. Fig. 35 shows the typical course of the changes of irritability in a case of moderate severity. In the muscle (M) a fall of irritability (due to the degeneration in the nerve-endings) occurs simultaneously with that in the nerve-trunk (N), and the faradic excitability becomes extinct at the same time in both nerve and muscle. The fall in voltaic irritability is quickly arrested by the change in the muscular fibres, through which they soon become more excitable than normal to the voltaic current. This change usually occurs during the second week, and the irritability continues to increase during the third and fourth weeks. At its maximum it may amount to three, four, or five cells of the battery, *i. e.* contraction can be obtained in the paralysed muscles with a current weaker by so many cells than is necessary to cause contraction in a corresponding unaffected part. The further course of the changes in irritability depends on the severity of the lesion and the intensity of the degeneration. In a case of moderate degree, such as is shown on the chart, nerve irritability reappears about the end of the second month, usually after some voluntary power is regained. It is at first low, so that a strong current is required. It gradually increases, but for a long time continues a little below the normal degree. This return of nerve irritability is accompanied by a corresponding return of faradic irritability in the muscles (*i. e.* in the intra-muscular nerves). The increase of voltaic irritability often persists long after recovery of power, but it lessens as faradic irritability returns, and, as shown in the chart, it may fall below the normal before it ultimately regains its original degree.

Slight changes in irritability can be ascertained only by a comparison with the corresponding part on the other side in the same individual. Moreover, when we speak of excitability being "lost," we mean that we can obtain no stimulation by any strength of current that can be borne. The sensitiveness of the skin varies in different persons and in different parts. The resistance of the skin also varies; the greater it is the stronger must be the current used to secure the action on the nerves below. Hence the importance of ascertaining what current is actually passing by the use of a galvanometer. It may be used once to gain information as to the significance of the number of cells that constitutes the evidence of the difference between the two sides when the sensitiveness to the pain which is caused prevents the use of the instrument in all measurements.

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* For a careful analysis of abnormal electrical reactions in muscle and nerve see Doumer, 'Bulletin Offic. de la Soc. franç. d'Electrotherapie,' 1897. The author lays especial stress on the importance of recording the nature of the excitation and the character of the muscle-curve produced.

If the lesion is very severe, so that there is no recovery, and no regeneration of the nerve, the loss of nerve irritability, and of the faradic muscular irritability, is permanent. The increase in voltaic irritability persists for months, and then gradually falls as the muscular fibres waste, and becomes lower and lower (see Fig. 36), until ultimately no reaction can be obtained, the fibres having perished. It does not become extinct until at least a year has elapsed, and sometimes (as in the chart) only towards the end of the second year. Often, when no contraction can be produced on the first attempt, after two or three applications, distinct contractions are obtained.

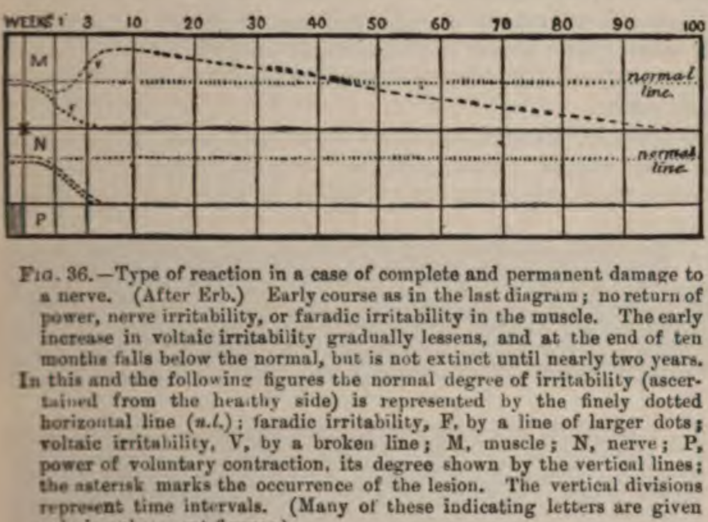

FIG. 36.—Type of reaction in a case of complete and permanent damage to a nerve. (After Erb.) Early course as in the last diagram; no return of power, nerve irritability, or faradic irritability in the muscle. The early increase in voltaic irritability gradually lessens, and at the end of ten months falls below the normal, but is not extinct until nearly two years. In this and the following figures the normal degree of irritability (ascertained from the healthy side) is represented by the finely dotted horizontal line (*n.l.*); faradic irritability, F, by a line of larger dots; voltaic irritability, V, by a broken line; M, muscle; N, nerve; P, power of voluntary contraction, its degree shown by the vertical lines; the asterisk marks the occurrence of the lesion. The vertical divisions represent time intervals. (Many of these indicating letters are given only in subsequent figures.)

The changes we have hitherto considered are in the *degree* of irritability, "quantitative" changes. But the quantitative increase in the muscular irritability is often accompanied by a change in the order of response, according to the pole that is applied and the strength of current—a "qualitative" or "polar" change. We have already considered its general characters (p. 31), and have seen that it consists in an undue readiness of response at the positive pole (anode) compared with that at the negative pole (kathode) (Fig. 37, B and C), the muscle being normally the more sensitive to the latter (Fig. 37, A). Writing Cl. for the closure of the circuit, O. for its opening, and C. for contraction, the normal reaction is—

1. K.Cl.C.; 2. $\begin{cases}\text{An.Cl.C.;}\\ \text{An.O.C.;}\end{cases}$ 3. K.O.C.

In disease—

1. $\begin{cases}\text{K.Cl.C.;}\\ \text{An.Cl.C.;}\end{cases}$ 2. An.O.C.; 3. K.O.C.: or even

1. An.Cl.C.; 2. K.Cl.C.; 3. K.O.C.; 4. An.O.C.

This qualitative change is at times absent, especially in cases of neuritis. It may be slight, and only amount to an equal kathodal and anodal excitability. It is practically constant when a nerve has been actually destroyed. On the other hand, it may be present and significant where less acute disease has not yet caused obtrusive wasting. Even when there is a marked quantitative change, the kathodal closure contraction may still occur first. When the change is present it is only in the muscles, and it must depend on the muscular fibres themselves. In the motor nerve the kathodal response is always the first, although a qualitative change has been detected in degenerated sensory nerves.

The muscular contractions which occur thus with undue readiness differ from normal contractions, excited through the nerves, in their distinctly deliberate character. Instead of the quick, lightning-like contraction, the movement is distinctly longer in reaching its maximum and longer in its duration. The recognition of this feature is easy, and often of practical importance when there is doubt whether the

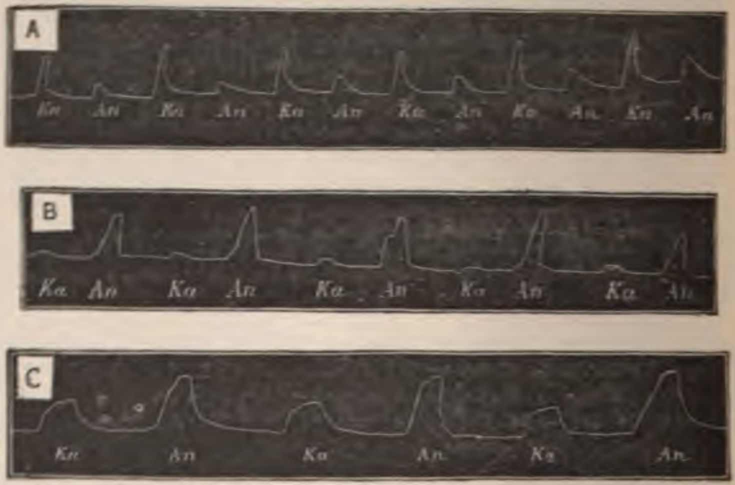

FIG. 37.—Tracings of the muscular contractions in nerve-degeneration. (After Erb.) *Ka*, kathodal closure contraction; *An*, anodal closure contraction. A, tracing in health; *Ka* much stronger than *An*; contractions sudden. B, tracing in nerve-degeneration with moderate current. *An* much greater than *Ka*, the latter scarcely visible. Contraction slower, shown by the more sloping upstroke. C, the same, with a stronger current. *Ka* greater, but still less than *An*; the slow character of the contraction and its long duration well marked.

voltaic current is acting through the nerve-twigs, or on the muscular fibres themselves. Sometimes a slight abnormal tetanic contraction during the passage of the current also occurs. During the period of increased voltaic irritability the mechanical excitability of the fibres is often increased. If they are directly percussed, they respond with a distinct slow contraction (Erb).

Such are the changes in irritability which occur in cases of nerve-lesion and degeneration of moderate and considerable degrees of severity. Certain variations are occasionally met with, and these occur especially in cases of neuritis of slight degree. In severe cases the fall in nerve irritability, which usually commences in the middle of the first week, may not take place until the end of this week, although it may then progress so rapidly that no response to stimulation can be obtained at the end of the second week. An example of this is shown in Fig. 38.* The same chart, and some of those that follow it, illustrate another very common variation from the type above described. There may be no fall in the voltaic excitability of the muscles before the commencement of the degenerative increase. Indeed, this initial fall is as often absent as present. The change in the nutrition of the muscle may then coincide with, instead of succeeding, the degeneration of the nerve-endings (Figs. 38, 40). Sometimes, if the degeneration is rapid, there is an interval of a few days before the secondary changes in the muscle have reached the degree necessary to produce increased irritability. In a slight degree of degeneration the increase in the voltaic irritability of the muscles may be postponed for a week or more after the nerve irritability begins to fall, and until the rise occurs the

FIG. 38.

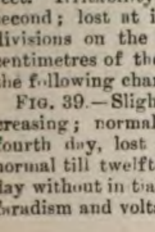

FIG. 39.

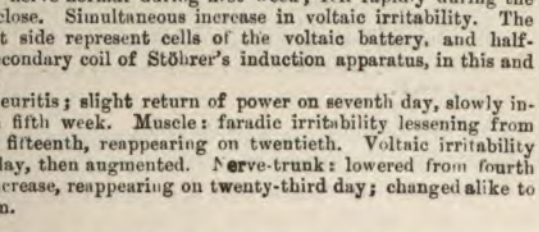

FIG. 38.—Severe neuritis, paralysis complete for months; recovery imperfect. Irritability of nerve normal during first week; fell rapidly during the second; lost at its close. Simultaneous increase in voltaic irritability. The divisions on the left side represent cells of the voltaic battery, and half-centimetres of the secondary coil of Stöhrer's induction apparatus, in this and the following charts.

FIG. 39.—Slight neuritis; slight return of power on seventh day, slowly increasing; normal in fifth week. Muscle: faradic irritability lessening from fourth day, lost on fifteenth, reappearing on twentieth. Voltaic irritability normal till twelfth day, then augmented. Nerve-trunk: lowered from fourth day without initial increase, reappearing on twenty-third day; changed alike to faradism and voltaism.

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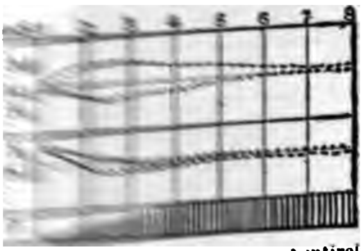
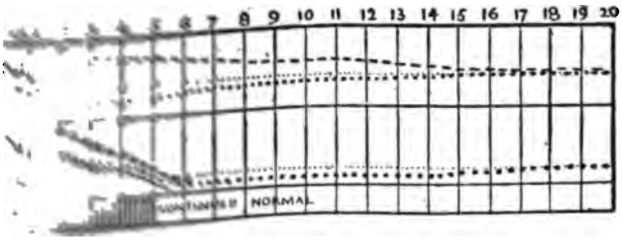
* This and the following charts are from cases of neuritis of the facial nerve.

[illegible] of the muscles may remain normal, even when their [illegible] falls with that of the nerve-trunk. An instance of [illegible] Fig. 39.



[illegible] not entirely [illegible] during the [illegible] in muscle [illegible] week, and con- [illegible] Nerve irritability [illegible] week, passing [illegible] depres- [illegible] falling more [illegible]

[illegible] lesion is very slight in degree, the fall in nerve irritability [illegible] by a rise, which may be far greater in degree than the subsequent fall. In slight cases the fall may indeed be not only slight, it may be altogether absent, so that the rise constitutes the only symptom. This initial increase in irritability is important as the manifestation of the slightest degree of alteration in the nutrition of nerve-fibres.* It may last for a few days or for two weeks (Fig. 40), and I have once known it to continue for five weeks (Fig. 41). Although the change in irritability of the nerve is usually the same to both currents, a [illegible] this rule is presented by the increase in irritability [illegible]. The change is not always quite equal to [illegible] voltaism, and it is frequently much more marked to [illegible] shocks than it is to either the voltaic current or the [illegible] shocks that constitutes the faradic current. This [illegible] in Fig. 41, and in less degree in Fig. 43. In [illegible] irritability to the two currents was the same; in the [illegible] voltaism was distinctly the greater. I have once met [illegible] distinct and prolonged diminution in faradic irritability [illegible] could be found to voltaism (see Fig. 42).



[illegible]: quick recovery of power during third and fourth [illegible] in muscles present at the end of the second week, [illegible] present in slight degree until the fourth month. [illegible] in irritability in the nerve-trunk at the end of the [illegible] to the faradic shock (crosses), and lasting until the [illegible]

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* [illegible] met with in some central diseases, as chorea and paralysis

Lessened irritability to faradism with distinct increase to voltaism has been observed in an ulnar nerve, the seat of traumatic paralysis (Bernhardt). In one case of extensive peripheral neuritis of obscure toxic origin, in which the face on both sides was paralysed for a time—a peculiar nutritional change in the nerves, where the action of the toxic agent was less than at their extremities, and made the nerves insensitive to the momentary induced current,—the voltaic excitability of the facial nerves was not lessened, although faradic irritability was lost to such a current as could be borne.

FIG. 42.

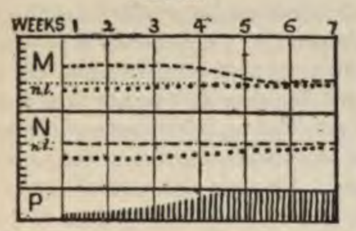

FIG. 43.

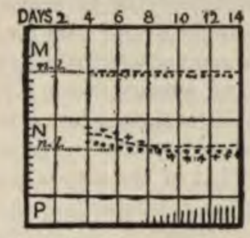

FIG. 42.—Slight neuritis: power regained during the second week. Deg. react. in muscle distinct on eighth day, lessening during second and third weeks. Nerve: lowered faradic, normal voltaic irritability.

FIG. 43.—Very slight neuritis. No change in muscular irritability; irritability of nerve-trunk increased from fourth to eighth day, most to faradic shock (crosses), least to faradic current.

The electrical excitability of a nerve and its power of conduction are usually lost together. But when a nerve is recovering from a local lesion, and regeneration is in progress below, some power of conducting the impulse that is excited above the lesion may return in the peripheral portion before it becomes excitable by any form of electricity. This is true of the impulse from the brain, and that excited by electricity. This seems to show that the nerve-fibres can conduct—that is, pass on the nerve-force they receive as such, before they can produce nerve-energy in response to the influence of a "stimulating" force acting on them. Another rare anomalous condition has been noted by Bernhardt and Grünberg in cases of recent nerve injury. When the lesion arrests all conduction of the voluntary stimulus a strong faradic stimulation of the nerve above the lesion may still cause slight contraction in the paralysed muscles.

Recovery of the nerve is attended by gradual restoration of power over the muscles, the nutrition of which slowly improves; but if the wasting has been considerable, a long time elapses before they regain their normal size. Indeed, as already stated, they may be always somewhat smaller than the corresponding muscles on the unaffected side. Curious secondary spasmodic symptoms are common after palsy of the facial nerve (*q. v.*)—contractures, spontaneous contractions, and associated over-action of different muscles. The last is very rarely met with after palsy of the nerves of the limbs, but coarse, fibrillary, muscular contractions, and a tendency to cramp, are not uncommon, and may persist for years. (See also Neuritis.)

Sensory symptoms also result from lesions of the nerves that contain sensory fibres. The interruption of the fibres arrests conduction, and causes loss of sensation in the part supplied by the nerve, just as it causes motor palsy. But a slight lesion of a mixed nerve may cause persistent muscular paralysis, and sensation be unaffected, or impaired only in slight degree and for a short time. This result is so frequent that we cannot ascribe it to a difference in the relative damage to the motor and sensory fibres. It must be due either to the fact that the sensory fibres recover more readily, or that a slight degree of conduction may suffice for the stimulation of the sensory centres in the brain, and not for that of the muscular fibres. There is also a third hypothesis, and that is a difference in the sensory and motor impulses in physiological character.* In another class of cases, however, we must seek a different explanation of the persistence of sensibility in the area supplied by the nerve. There may be no loss even when a nerve is completely divided. This is observed chiefly near the extremity of a limb. It can only be explained by what is termed "recurrent sensibility." Anastomoses exist between the terminal fibres of different nerve-trunks, and it would seem that this contiguity of sensory fibres suffices for conduction. Individual differences are seen to exist in this. Thus, of two persons in whom the ulnar nerve at the wrist has been divided, one may have total anæsthesia in the fingers supplied by the nerve, and in the other there may be little or no loss.

The peripheral segments of the sensory fibres degenerate, like the motor nerves. We cannot test them, and ascertain the degeneration in the same way, at least in severe lesions, because their stimulation cannot be perceived. Nevertheless an altered polar reaction has been observed in slight cases, analogous to that which occurs in the muscles. A sensation is produced with the positive voltaic pole by a weaker current than is required with the negative pole. In some cases of nerve injury and neuritis a delay in conduction of pain may be observed.

Increased and disordered sensitiveness in the area of distribution of the nerve, with or without spontaneous pain, is very common in cases of partial lesions of nerves. It must be referred to the morbid changes in their fibres, alterations in the nutrition of the axis-cylinder or nerve-endings, probably analogous to the slight changes in the motor nerves which increase their excitability. Pain and tenderness of the nerve-trunk are also frequent in the same cases, due no doubt to the increased sensitiveness of the "nervi nervorum" distributed in the sheath. The sheath of each nerve is part of the distribution of its own fibres, and those that end in the sheath seem to be very readily deranged. We shall see the importance of this consideration in relation to neuritis and

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he experiments of Leuderitz ('Zeitschr. f. klin. Med.,' Bd. iii) merely confirm rved fact, and do not really explain it, as he and others have assumed.

neuralgia. The pains, both local and distant, are sometimes very severe and persistent.

Reflex Action.—All lesions of nerves abolish reflex action in the area of the distribution of the nerve affected, except those that involve so slight a change as to cause increased sensitiveness. Myotatic irritability is also lost, from damage to either the motor or sensory fibres concerned, and this loss may persist long after the other symptoms.

Vaso-motor and trophic disturbance is a common consequence of lesions of nerves. It is most severe in those that are inflammatory in part or altogether. The vaso-motor nerves run in the mixed nerve-trunks, and suffer with the other fibres. The general character of the disturbance has already been described (p. 27). When the lesion is acute and irritative, vascular dilatation and an increase of temperature occur at first, and are followed by passive hyperæmia and lowered temperature. There may be œdema, and an increased secretion of sweat. The changes in the skin, acute and chronic, are those that have been already described. "Glossy skin," red, smooth, and thin, is especially common, and is frequently accompanied by atrophy of the subcutaneous tissue, and perhaps of the bone, so that the finger-tips become narrow, and present an aspect that is very characteristic. Burning pain, with tenderness, often accompanies the change, and may continue for long. Occasionally, instead of becoming thin, the epidermis increases in thickness, the nails often become thick and rough, and present transverse or longitudinal furrows.* In parts where there is much subcutaneous tissue this may also become thickened and have a peculiar doughy feel, resembling that met with in myxœdema. The growth of the hair may be lessened or increased. Sloughing of the skin is far less common than in central diseases, but eruptions, particularly of the nature of erythema, eczema, herpes and pemphigus, may also be met with. Vesication occurs with extreme readiness, and sometimes vesicles or bullæ appear to form spontaneously. Ulcers result which erode and destroy the tissues, especially of the finger-ends. Mustard plasters also blister the skin more readily than in health. It is important to remember these facts, because the affection is one in which hot applications are often recommended. A gentleman dislocated his shoulder, and either by the displaced bone, or in the reduction, the brachial plexus was seriously injured. The muscles of the hand wasted, and the skin became glossy. He was advised by a surgeon to bathe the hand daily in hot water. One day his wife bathed it in water which to her was pleasantly warm, and which caused him no discomfort, but the result was that the hand was covered with blisters, and ulcers were left which did not heal for months.

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* Several excellent illustrations of these changes are given by Bowlby, loc. cit., where also a full account of traumatic lesions is given. The observations of Bernhardt ('Virchow's Archiv,' 1886) appear to show that the retarded growth of the nails is independent of the influence of the particular nerves. Great individual variations are found with identical lesions.

The joints often suffer in their nutrition. Acute inflammation, such as occurs sometimes in spinal affections, is rare, but chronic changes in the joints are frequent, with much tenderness; alterations in the articular surfaces, and fibrous adhesions result, which limit movement. When the muscular wasting is slight, the condition may resemble a primary joint affection, and this mistake in diagnosis is sometimes actually made.

INJURIES OF NERVES: COMPRESSION.

Traumatic lesions of nerves are in the province of surgery. They belong to the physician only in so far as their effects are produced by the agency of a neuritis, the mechanical origin of which may escape notice. Neuritis is described in the next section. A word is needed, however, on the subject of simple, slow compression of nerves, not because the symptoms are grave, but because they often give rise to very grave mental concern on the part of the patient, and to occasional doubts in the mind of his medical attendant. Compression of a nerve may be sudden or slow. Sudden compression bruises the nerve-fibres, producing definite lesions, and also sets up neuritis. Symptoms often occur too soon to be explicable as neuritic. A slight degree of these is familiar as a result of pressure on the sciatic nerve in the act of sitting. There is first tingling in the region of distribution of its fibres, often with a sense of warmth, with disordered sensations and even a loss of sensibility in the skin. Often this is accompanied by an inability properly to put in action the muscles that are supplied by the nerve. All the symptoms pass away in a few minutes, a sense of cold and an inability to direct movements being often the last effects. If they do not pass away, the persistent effects are ascribed to a neuritis such as is produced by any other cause.

But neuritis is not the primary lesion, and the initial symptoms are not produced by the agency of inflammation. Hence, when they persist, it is doubtful whether we should ascribe them to the inflammation unless they occur after an interval. The mechanism by which the tingling and early anæsthesia are produced, probably causes also the lasting symptoms. The compression no doubt causes anæmia, but the known sensitiveness of nerves to mechanical stimulation makes it probable that the effects are chiefly produced by the mechanical influence of the pressure on the elements of the nerve-fibres, and that a compression for a few hours has such an effect in separating the molecular elements of the white substance, as to set up a secondary degeneration of the same character as that which results from division of a nerve. The neuritis is thus no more the cause of symptoms in compression than in division. In each there is a displacement of molecules interrupting conduction. This is also the conclusion of Weir Mitchell, who found that a pressure of eighteen to twenty inches of mercury for twenty seconds abolished the power of conduction, though only for a few minutes, in spite of conspicuous change in the white substance. The latter is still greater, and the effects more enduring, in a contusion.

The cause of the tingling in the region of distribution of the sciatic nerve is, as already said, readily recognised when it is due to pressure by sitting. But it is otherwise when the pressure is on the nerves of the arm and is produced during sleep. The tingling may be felt in the region of the ulnar nerve, in consequence of flexion of the elbow, or in the whole hand, from the pressure on the nerves of the brachial plexus on the side on which the patient is lying. Waking up with the tingling, and sometimes with cramp or powerlessness in the hand, he is naturally alarmed. The diagnosis, however, is rendered easy by the unilateral character of the symptoms, and by the fact that they are always felt on the side that is bearing the weight of the body. There may, however, be great difficulty in diagnosing this form of compression from actual contusions. The symptoms are produced much more readily when the general health is bad; they last longer, and more often end in neuritis.

Gradual compression may result from continuous pressure, such as that of a crutch or the growth of an adjacent tumour, or from the implication of the nerve in a cicatrix or callus. Of new growths compressing nerves, exostoses and aneurisms are most frequent. The symptoms of this form approach those of neuritis; they may be sensory, motor or trophic. They are ushered in by hyperæsthesia, which is followed by anæsthesia, seldom complete in degree or quality, tactile and muscular sensibility being usually retained when other forms are lost. Intense and persistent hyperæsthesia or complete anæsthesia point to neuritis. Motor weakness and trophic changes appear; there may be atrophy or hypertrophy of the nails, trophic ulcers, or joint affections. Among rarer symptoms are local spasm with fibrillary twitchings and reflex convulsions. In simple compression the symptoms, as a rule, disappear after the removal of the cause; the subjective sensations are the first to vanish, then the anæsthesia goes, and lastly—in severe cases not till after some months—the motor disturbances.

In rapid compression there may be, according to the duration of the injury, merely congestion at the affected spot or peripheral degeneration starting from it. A nerve which has been long compressed is narrow at the affected part, where it may even be flattened into a ribbon; above and below it is normal or swollen. The compressed portion may be white from anæmia or red from congestion. Eventually degenerative or neuritic changes may develop, and the former are sometimes found above the lesion as well as below.

Treatment is essentially surgical, but the physician must aim at maintaining the nutrition of the muscles by the usual means during the recovery of the nerve, when this can be released from compression.

INFLAMMATION OF NERVES: NEURITIS.

Neuritis, or inflammation of nerves, presents various characters, which have led to the distinction of numerous forms. The inflammation of single nerves usually begins in the outer sheath of the nerve, and this has been distinguished as "perineuritis." It may involve also or chiefly the connective tissue between the bundles of nerve-fibres ("interstitial neuritis"), or the nerve-fibres themselves ("parenchymatous or degenerative neuritis"). Some forms of neuritis have a tendency to ascend the nerve, and this variety has been termed "ascending neuritis" or "neuritis migrans." One nerve-trunk may be primarily affected, or many nerves may suffer at the same time. The latter condition, "primary multiple neuritis," is of great importance, and will be described in a separate section.

Most forms of neuritis may be either acute or chronic. Acute neuritis usually subsides into a chronic stage, and its symptoms may thus persist for a long time. Other varieties that have been distinguished depend upon the cause to which the inflammation is secondary. Thus we have cancerous, syphilitic, gouty, diabetic, and other forms.

Causes.—Injury causes more or less inflammation, which occurs readily, and follows very slight damage. Neuritis may thus be set up by all sorts of wounds, by contusions, by compression from without or by adjacent structures, and by over-extension of the nerves. Those nerves that pass by joints are liable to injury from dislocations, either by the displacement of the bones, or during reduction. In fractures the nerves may be injured directly, or may be compressed by the callus that is formed. Nerves are sometimes damaged by a violent contraction of the muscles through which they pass. It is probable also that muscular effort excites neuritis in other situations, especially in persons who are predisposed. Moreover, a strain on the fibrous tissues of joints, fasciæ, &c., may set up inflammation in them, which may spread to the nerves.

Neuritis may arise by extension from adjacent inflammation; this is particularly seen in the case of phlebitis. The nerves near suppurating joints may be involved, and even, it is said, those that pass by a joint the seat of simple inflammation. The latter is certainly rare. The intercostal nerves have been affected by extension from an inflamed pleura. The cranial nerves and the spinal nerve-roots are involved by extension from the membranes.

Exposure to cold is another cause of neuritis. It is then often called "rheumatic." The nerve, or rather its sheath, may be primarily affected, or the first effect may be inflammation of the fasciæ, from which the inflammation spreads to the nerve-sheath. Persons who are liable to muscular rheumatism, and those who are gouty, suffer thus with especial frequency. But exposure to cold and the toxins of gout and rheumatism also act in another way, by causing a blood-state which acts on many nerves at the same time. Many general diseases have a similar power of causing either an isolated or a multiple neuritis. It is through this agency that diphtheritic paralysis is produced, and the same effect may be due to the presence of other toxic material in the blood, metallic poisons, alcohol, &c., an account of which will be found in the section on Multiple Neuritis. When an isolated neuritis occurs during an acute disease it is probably produced through the agency of compression, unnoticed during the prostration. Neuritis has also been found in the vicinity of bedsores (Pitres), and though probably due to pressure may be part of the mechanism by which they are caused. Some chronic general diseases cause only isolated neuritis,—either simple, as that which is due to gout (when the general disease must be regarded chiefly as a predisponent); or special, as syphilitic, cancerous, or leucocythæmic neuritis, in which there is an infiltration of the nerve with the special tissue-element characteristic of the general disease.

The characters of the simple isolated form constitute the chief subject of this section.

Pathological Anatomy.—The changes differ according as the inflammation affects primarily the adventitial connective tissue, "adventitial neuritis," or the fibres themselves in the "parenchymatous" form. Isolated neuritis is generally adventitial, and will be first described. In acute inflammation the affected part of the nerve is red, softened, and swollen. The redness depends on distended vessels, which may be visible on the surface; in more acute cases it may result from the presence of minute hæmorrhages. The swelling is due to œdema, or to a sero-fibrinous exudation, sometimes jelly-like in aspect. The microscope shows leucocyte-like corpuscles surrounding the vessels, infiltrating the sheath (Fig. 44) and accumulating between it and the nerve. There may be even small extravasations of blood. Suppuration is extremely rare. These changes may be limited to the sheath in what is called "perineuritis," or may extend into the substance of the nerve in "interstitial neuritis." In the latter case the lymphoid corpuscles infiltrate the septa, and may even be seen in the substance of the fasciculi, between the nerve-fibres. These changes

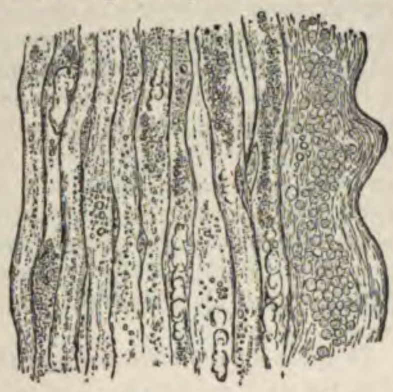

Fig. 44.—Neuritis: degeneration of nerve-fibres, the myelin broken up into masses, globules, and granules. Accumulation of leucocytes in nerve-sheath. From a case of multiple neuritis. (After Leyden.)

may be continuous along a considerable tract of the nerve (diffuse neuritis), but more frequently they are chiefly marked at certain places, which are separated by portions of the nerve which are so little affected as to appear to the naked eye to be normal ("focal" or "disseminated neuritis"). The foci of inflammation are chiefly situated at places where the nerve turns round a bone, or emerges from canals or fasciæ, or divides.

The extent to which the nerve-fibres suffer varies much. They usually present little change when the inflammation is limited to the sheath, unless the nerve lies in a bony canal, or in rigid fibrous tissue, within which the sheath cannot expand; its swelling then exerts pressure on the fibres. When the inflammation is interstitial the fibres suffer more readily, although not invariably. On the other hand, they are sometimes found much altered, when the connective-tissue elements are but little affected. In this case the inflammation is "parenchymatous" and begins in the nerve-fibres, the interstitial tissue being secondarily involved or unaffected. The changes in the fibres are almost the same as in degeneration (Figs. 33 and 34). It was pointed out how closely the two processes of inflammation and degeneration are connected, and how difficult it is to separate, or even to distinguish them, in many cases. The myelin of the white substance first breaks up into segments, more or less elongated, often with smaller globules of myelin between or beside them. The masses are cloudy or finely granular in aspect. The axis-cylinders are interrupted where the myelin is divided. The nuclei of the sheath are increased in number, and the protoplasm around them is increased in quantity. Next the myelin is divided into smaller globules and granules, and the axis-cylinder is no longer recognisable. The myelin then disappears in parts of the tubes, while it remains at other parts, but is still more finely divided. Lastly, the sheaths become empty and very narrow, containing only nuclei at intervals, with here and there a little finely granular material, or sometimes some brownish pigment granules. The process may thus go on to complete destruction of the fibres. These commonly suffer unequally; fibres that have a normal appearance are scattered among those that are much altered.

In chronic interstitial neuritis the axis-cylinders often suffer less than the white substance. The latter becomes atrophied, so that the fibres are smaller than normal. If the interstitial inflammation is very intense the fibres may break up as above described. As the inflammation subsides, the new cellular elements that have made their appearance assume the aspect of fusiform cells, and fibrous tissue is developed, either from these cells, which become less numerous, or from the intercellular exudation. This fibrous tissue surrounds and encloses the fasciculi, a condition that has been termed "sclerosis of the nerve." A firm fusiform swelling may remain at the affected spot, and this may be adherent to adjacent structures. The axis-cylinders are unequally affected. Those which are involved undergo typical degeneration. Sometimes fat is ultimately formed in the new connective tissue, a condition that has been rather unnecessarily termed "lipomatous neuritis" (Leyden); the fatty deposit does not seem to be related to any special form of neuritis. Occasionally the connective tissue overgrowth is so extensive that the whole nerve is converted into a sclerotic cord (Virchow's "proliferative" form).

In syphilitic neuritis, which affects chiefly the cranial nerves, there is a cellular growth in the sheath and interstitial tissue similar to that which constitutes other syphilitic new formations, with a variable amount of alteration of simpler inflammatory character. The growth in the sheath may amount to a distinct syphilitic tumour, with or without interstitial changes. In cancer, nerves adjacent to the new growth may present simple interstitial neuritis, or may be infiltrated by a growth of cancer-elements spreading to the nerve by direct extension. In leucocythæmia there is a dense infiltration of the nerve with leucocytes.

The destructive changes in the nerve-fibres extend down the nerve to the periphery in a process of secondary degeneration, more or less inflammatory in nature, as already described in secondary degeneration. Usually the central portion of the nerve remains free, the alterations ceasing a short distance above the seat of the primary inflammation. In rare cases an ascending neuritis (n. migrans) passes up the nerve, and may spread, at a plexus, to other nerve-bundles and other nerve-trunks. The formation of fibrous tissue in the inflamed part, and the cicatricial contraction of this tissue, often prolong the irritation of the nerve-fibres and the symptoms due to this, and a state of "chronic neuritis" is said to be left.

SYMPTOMS.—The symptoms of neuritis vary extremely according to its intensity, its extent, and the nerve that is affected. In three places, the brachial plexus, the sciatic nerve, and the crural nerves, the symptoms are sufficiently special to make their separate description desirable, although the general account applies to them also.

The onset of the acute form is sometimes attended by some constitutional disturbance, especially when many nerves are affected. The chief symptoms are local. The most prominent is pain, felt in the inflamed part of the nerve, and also often in the part to which it is distributed (see p. 78). Sometimes the pain involves the whole limb, and in severe cases it may be most intense, burning, boring, rarely darting, in character; it is usually worse at night and increased by movement, by postures that involve tension or pressure on the nerve, and by whatever causes passive congestion of the limb, such as the act of coughing. Sometimes it seems to radiate into distant parts, and not rarely pain is also felt in the corresponding region of the opposite limb. The sensitiveness of the whole of the affected region is increased, and even the bone may be tender, so that at first attention may not be directed to the nerve, but when this is pressed great pain is always produced. In slighter cases the pain is usually limited to the nerve and its distribution. It may, indeed, be absent when the nerve or branch is chiefly motor, a fact difficult to explain unless the sheaths of such nerves contain fewer sensory fibres than usual. If the nerve is accessible to direct examination it may be felt to be distinctly swollen at the affected part. Occasionally the skin over it has been observed to be red, and rarely there has been slight œdema. Spontaneous sensations may be felt in the region supplied by the nerve, tingling, &c., and the skin may be hyperæsthetic. After a time, as the nerve-fibres suffer, sensation may be perverted, or even lessened; complete anæsthesia is usually confined to small areas. The muscles supplied by the nerve become weak in various degree, tender, and present fibrillar twitchings; they are seldom powerless, but pain may prevent their contraction. They present the altered electrical reactions characteristic of nerve-lesions. Increased perspiration has been observed in the part of the skin supplied by the inflamed nerve, very rarely it is arrested, and sometimes, although rarely, eruptions occur. Herpes is not common from ordinary acute neuritis. The epidermis may become thickened into a state like ichthyosis, or may atrophy and appear as "glossy skin." Effusion into joints has been observed in very rare cases. The constitutional disturbance which may attend the onset subsides in the course of a few days, but the pain and other symptoms usually persist in undiminished severity for several weeks. They ultimately slowly subside into a chronic stage, and may continue as a secondary neuralgia due to a habit of over-action set up in the centre.

In the chronic form constitutional disturbance is absent, and pain is the prominent symptom from the first. The affection of motion and sensibility, and the trophic changes, are similar to those met with in the acute form. In both forms the muscles may waste, and present the reaction of degeneration when they are tested with electricity (p. 73). In slight cases the initial increase of excitability in the nerve is often well marked. Trophic changes in the skin are very common; so is alteration in the nutrition of the joints, and adhesions form, limiting the movement, and fixing the parts in the position corresponding to the muscular inaction.

When neuritis ascends a nerve, the symptoms gradually extend in area; and if it reaches the plexus from which the nerve proceeds, they may extend to most or all the nerves of the limb. This "ascending neuritis" is not rare in man, may even reach the spinal canal, and there produce various disturbances. The inflammation may spread in the tissue outside the dura mater, or may extend to the cord, and cause subacute or chronic myelitis with or without meningitis. The paralysis that occurs secondarily to some visceral diseases, as those of the bladder, and commonly regarded as reflex paralysis, is probably produced through the agency of an ascending neuritis. Lastly, the inflammation may extend to the nerves of the other side, usually to those that correspond to the primary seat of the disease. Such extension may be through the spinal cord or the membranes, but in some cases symptoms occur in the nerve of the opposite side, without any indication of an affection of the centres. Experiments on animals have demonstrated that such implication of the opposite nerve may occur when the centres are unaffected. It has been called "sympathetic neuritis." We have seen that reflected pains are occasionally felt in the corresponding nerve on the opposite side, and it is probable that, as in neuralgia, vaso-motor disturbance in the nerve-sheath may accompany such pains, and, in a predisposed person, may cause actual neuritis.

The *Duration and Course* of neuritis vary much. A slight acute neuritis may run its course in a few weeks and then subside. Much more commonly the affection persists in a chronic stage for many weeks, or even months, and slowly passes off. The rheumatic and gouty forms are as a rule much more tedious than those that result from injury, although traumatic neuritis is sometimes very insidious and may last a long time; it may ascend the nerve from its starting-place and become localised in certain situations, where it may develop afresh, and give rise to symptoms that often seem to be independent of their cause. In the predisposed, it can set up troublesome local functional disturbance, hysterical contracture, and the like.

DIAGNOSIS.—The diagnosis of neuritis depends, first on the localisation of the symptoms to the distribution of a certain nerve-trunk, and secondly on the pain and tenderness in the nerve. The diffuse pains that attend the onset may be readily mistaken for the pains of acute rheumatism, or for those due to an inflammation of the bone, but in the course of a day or two the localisation of the symptoms declares their nature. The chronic form is easily and often mistaken for neuralgia, and the diagnosis is the more difficult because many so-called neuralgias are really due to neuritis. The distinction can only be fully discussed when we have considered the symptoms of neuralgia, but it rests chiefly on the fact that in neuralgia the pain intermits more completely than in neuritis, there is not the same initial tenderness in the nerve-trunks, and the tender spots have a more uniform localisation. Lessened sensibility or change in the muscles, showing organic damage to the nerve-fibres, are conclusive evidence of neuritis. The pains in some central diseases, chiefly those of the spinal cord, may be thought to be due to neuritis, but there is not the local tenderness of the nerve-trunk, and the pain is not limited to the distribution of a single nerve. As we shall see presently, the diagnosis of multiple neuritis from affections of the cord is far more difficult.

PROGNOSIS.—The gravest form of simple neuritis is that in which the nerve is affected secondarily to a local suppurative inflammation. The prognosis is best, as a rule, in traumatic neuritis, but the rule is one to which the exceptions are occasionally very considerable in degree. A neuropathic disposition makes the prognosis worse. In all cases the intensity of the symptoms, and the evidence of descending degeneration, furnish a more trustworthy guide to prognosis than does the mere form of the affection. The effects of complete degeneration always endure for some months. Regeneration does not occur until the original cause has ceased to act, and then occupies many weeks in its progress to the restoration of functional competence. In all forms pain is apt to linger on, and the longer the older the patient is.

TREATMENT.—The first consideration in the treatment of neuritis is the removal of its cause, if this can be discovered. A wound or injury or local inflammation adjacent to the nerve must be dealt with by appropriate measures. Any constitutional cause, such as gout, must be treated. For the inflammation of the nerve, it is of the first importance to secure to the part as perfect rest as possible. Movement causes mechanical irritation of the nerve, and involves functional stimulation of its fibres, both of which are injurious. The nerves of a limb run between muscles, and the contraction of these muscles causes pressure on the inflamed nerve, and irritates it, as the pain thus produced sufficiently shows. Pain is indeed a useful indication of the harmful influence of movement, since the stimulation of the nerves of the sheath, in which the pain is produced, doubtless has also a reflex effect, increasing the vaso-motor element in the inflammation. Hence it is most important that all movement that causes pain should be avoided. The posture of the limb should be such as to involve the least pain and the least danger of secondary contracture. The general treatment of an acute neuritis must be that suitable for any acute local inflammation, whatever its seat,—an unstimulating diet, an aperient, and diuretics. In gouty cases a brisk purgative may, with advantage, be given. General diaphoresis is useful in cases that are due to cold, and in others local sweating is often of distinct service. The limb may be steamed, or exposed to hot air. This should be followed by hot fomentations applied along the course of the affected nerve. and these by linseed-meal poultices. Leeches may also be employed at the onset of severe cases. When the inflammation results from injury, cold may be applied along the course of the nerve instead of heat; both probably modify the vascular disturbance of inflammation in a similar manner. Counter-irritation may be used at the onset of slight cases; but when the inflammation is severe, this agent is more effective during the subsidence of the inflammation than during its active stage. Blisters, repeated mustard plasters, or stimulating liniments may be used, but care must be taken not to blister skin that is anæsthetic, or troublesome ulceration may be caused. The same caution is necessary in regard to hot applications, as is shown by the case mentioned on p. 79.

Spontaneous pain requires sedatives, of which the hypodermic injection of morphia is by far the most potent. It should be used only for spontaneous pain, and not to enable the patient to use the limb in a way that would produce pain if morphia were not given. Mechanical irritation may be equally injurious, although the pain which it would cause is obviated by the sedative. Moreover it should only be used as a last resort, when the agent to be mentioned next is inadequate. Morphia has little influence on the morbid process; it lessens the effect of the disease on the brain only by acting on the brain.

In cocaine we have an agent capable to a large extent of replacing morphia, and of a greater therapeutic capacity. Although it has less power of relieving pain, it is capable of doing much more than that. It arrests the local transmission of the impulses that cause pain, and so, at a limited region, it does lessen pain and often remove it for a time, and the local character of its action enables it to exert a simultaneous influence on the morbid process. It prevents the irritative influence on the inflamed structures of the impressions that are felt as pain, which we have just considered; and the entire arrest of the influence, even though the region is small, seems to produce a much greater effect on the process than might be anticipated. It is instructive to note the opposite influence, revealed by the occasional redness of the skin and subcutaneous œdema, along the course of the inflamed nerve. The injection should be made at one of the seats of pain. In the rheumatic form salicylate of soda has been given, but is of doubtful value. Iodide of potassium sometimes seems to be useful, but no agent has so distinct an influence on the process of inflammation in the nerve as small doses of mercury. A grain of blue pill may be given once or twice a day; and if morphia has to be injected at the same time, the mercury is useful also in correcting the constipating influence of the morphia.

In the chronic stage or form, counter-irritation, by blisters or cautery, is of great value. So also is electricity, which has little influence during the acute stage. The voltaic current should be used. The positive electrode may be placed over the inflamed part of the nerve, or over the seat of pain, and kept there for ten minutes at a time, the strength of current being slight, such as the patient can just perceive. In very chronic cases a stronger current, sufficient to cause actual pain, is of service, applied in a similar manner, but for a shorter time. It has probably chiefly a counter-irritant influence, and faradism may be used in the same way. All painful impressions on the skin lessen, for a time, the nerve-pain.

The muscles supplied by the inflamed nerve may be left alone, unless their wasting is marked, or the degenerative reaction shows serious damage to the motor fibres. In cases of moderate severity they will recover when the nerve recovers. All that is desirable is that they should be gently rubbed once or twice a day. If the wasting is considerable, however, or there is degenerative reaction, they should be stimulated to gentle contraction by a weak, slowly interrupted voltaic current. They should on no account be faradised during the active stage of the affection, even if they act to faradism. The acute pain that the faradic stimulus causes, and the increased tenderness that lasts for hours afterwards, are evidence of its injurious effect.

In all cases attention to the general health is of great importance. Tonics are needed during the chronic stage. Change of air will sometimes remove, in a few weeks, symptoms that have previously been stationary for months.

The modifications of treatment that are rendered necessary by the position of the neuritis will be considered when we come to speak of the affections of the several nerves. The pathological varieties of neuritis only need special treatment in so far as they depend upon special causes, and this point, as already mentioned, must always be one of the first considerations.

MORBID GROWTHS IN NERVES: NEUROMA.

The term "neuroma" has been applied indiscriminately to all morbid growths situated on the peripheral nerves. The discovery, by Virchow, that many of these consist of a growth of nerve-fibres, while others consist of heterologous tissue such as constitutes morbid growths elsewhere, has led to the distinction of the former as "true neuromata" and the latter as "false neuromata," or "pseudo-neuromata," and even to the limitation of the term "neuroma" to a growth of nerve-tissue, whether in the central or peripheral nervous system, the heterologous growths being called by the names they bear in other situations, as "fibroma," "sarcoma," &c. The latter system of nomenclature, although certainly more consistent, has not become current in this country.

The true neuromata may consist of medullated or of non-medullated nerve-fibres, termed "myelinic" and "amyelinic" forms by Virchow. The latter were for a long time regarded as fibrous. The occurrence of ganglion-cells has been proved in only one or two instances. There is connective tissue between the nerve-fibres, which varies in amount and in character, and hence the firmness of these tumours also varies. This interstitial tissue may be so abundant as to constitute an intermediate form between the true and false varieties. This is probably the condition in most cases of multiple neuromata. The "false neuromata" may be of various nature, but fibrous growths, "fibromata," are far more common than any other kind. Myxoma occasionally occurs, the new mucoid tissue growing from the nerve-sheath. In one case* a myxo-fibroma as large as a goose egg involved the sheath, and pressed on the median nerve, a piece of which, when resected with the tumour, showed hyaline degeneration of the medullary sheath, the axis-cylinders persisting in a slightly swollen condition. Glioma is very rare on the peripheral nerves, although occasionally found on the auditory nerve; it should be remembered that this form of growth arises from the same embryonal tissue as true neuroma. Various forms of sarcoma have been met with. Carcinoma also occurs, very rarely as an isolated growth, but not uncommonly as a more or less diffused or nodular infiltration of the nerve, arising by extension from a contiguous growth.[*] Syphilitic growths are common on the cranial nerves within the skull, but are rare elsewhere. In lepra anæsthetica the nerves are infiltrated with fibrous tissue, enlarging them to many times their normal size. The enlargement is rarely nodular, and is rather a chronic cirrhotic inflammation than a growth. (See Leprous Neuritis.)

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* Schüster, 'Zeitschr. f. Heilkunde,' 1886, vii.

A curious variety of neuroma, consisting of interlacing cords, more or less nodular and tortuous, is termed *plexiform neuroma*. The disease commonly begins in fœtal life, and is most common on some branch of the fifth nerve in the orbit or the upper eyelid, but has been met with over the temporal bone, in the lumbar, cervical, brachial, and solar plexuses, on the penis and the mamma. It may be quite superficial or deeply seated. The cords, from 1 mm. to several centimetres in diameter, consist of a clear outer zone of concentric fibrillar connective tissue, of inner looser nucleated tissue, and, in the middle, a bundle of nerve-fibres, some normal, others degenerating. The cords are connected together by loose tissue, sometimes myxomatous, separated from the proper tissue of the cords by an epitheliated space. The growth of this form is extremely slow, but it may exert compression on adjacent structures.[†] A remarkable case of extensive myxomatous disease of the nerves of the forearm, congenital in origin, presenting analogies to plexiform neuroma, is recorded by Mr. De Morgan.[‡]

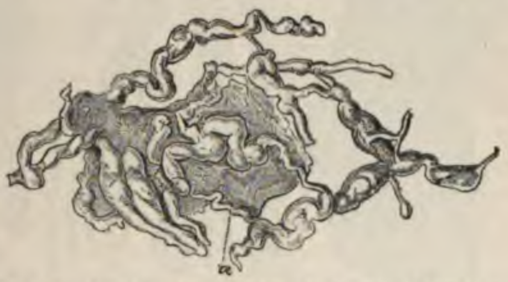

FIG. 45.—Plexiform neuroma from the orbit. (After Marchand.) The connective tissue surrounding the cords has been removed except at *a*, and irregular, cylindrical, nodular cords are seen anastomosing. A nerve passes into the tumour and suddenly becomes enlarged.

[*] A unique case of rhabdomyoma occurring in a nerve is recorded by Orlandi ('Arch. p. l. sc. med.,' xix, 5).

[†] Marchand, 'Virchow's Archiv,' Bd. lxxvi, p. 36.

[‡] 'Path. Trans.,' vol. xxvi, p. 2.

The subcutaneous extremities of sensory nerves are sometimes enlarged into minute tumours which, when painful, have been termed "tubercula dolorosa." Neuromata in the skin may co-exist with similar tumours on the nerve-trunks. Those shown in Fig. 46 are from the same case as the tumours represented in Figs. 47—49.

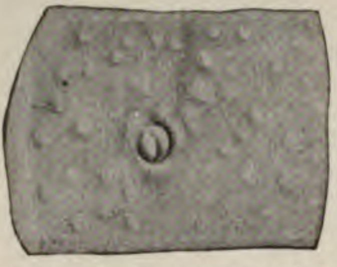

FIG. 46.—Cutaneous neuromata from skin of abdomen. (After Smith.)

The size attained by growths on nerves varies from that of a child's head to a nodule only just visible. They rarely exceed the dimensions of the closed fist. The variation in size of multiple neuromata is illustrated in Figs. 47—49, after Smith.* The large tumour (Fig. 47) was the size of a lemon, but in the same case a growth from the sciatic plexus almost filled the pelvis. They may occur on any nerve in the body, cranial or spinal, and are sometimes numerous on the pneumogastrics, spinal roots, and sympathetic plexuses. When neuromata are multiple

FIG. 47.

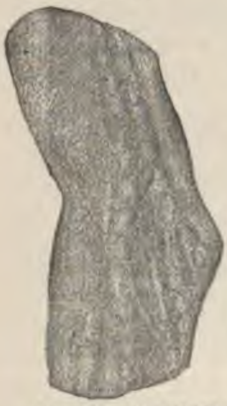

FIG. 48.

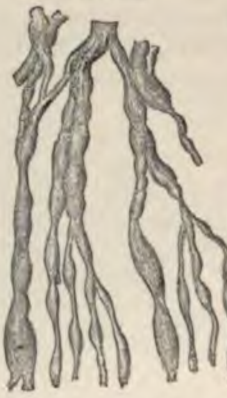

FIG. 49.

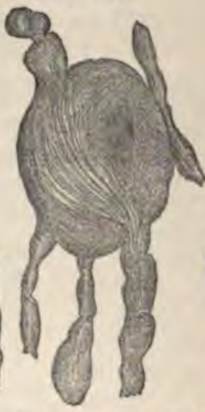

FIG. 50.

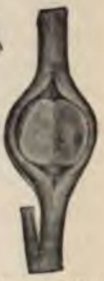

FIG. 47.—Right elbow of a man, æt. 30, with multiple neuromata; those on the nerves of the arm are visible beneath the skin as moniliform series of swellings along the course of the nerves. (After Smith.)

FIG. 48.—Part of the brachial plexus and nerves of the arm.

FIG. 49.—Neuroma of the posterior tibial nerve, same case. The tumour was the size of a lemon, and could be felt in the popliteal space. The flattened bundles of fibres of the nerve were separated and passed over the surface of the tumour. The portion of nerve to the right is a piece of the external popliteal. The tumours caused no interference with function. The patient died of enteric fever. One large tumour filled the pelvis.

FIG. 50.—Small neuroma of popliteal nerve laid open. It is seen to lie within the sheath. It caused no symptoms during life. (After Smith.)

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* R. W. Smith, 'Treatise on Neuroma,' 1849. Many instructive cases have been collected by Bowlby ('Diseases and Injuries of Nerves').

they are often extremely numerous, and almost every nerve in the body may be transformed into a chain of growths. In the case figured more than 200 were counted in the right arm alone, and the total number of growths in the body cannot have been less than 1000, while in another 3020 were counted.[*] These multiple neuro-fibromata have a great tendency to undergo sarcomatous degeneration. They have hence been termed "secondarily malignant neuromata."[†]

The tumours are almost always within the sheath of the nerve (Fig. 50). Sometimes they are on one side, and the nerve may pass by unchanged. More often the substance of the nerve is involved, and the fibres may be separated and spread out on the surface (Fig. 49). Even then they may not be damaged. They suffer far more in heterologous growths than in true neuromata

CAUSES.—The causes of neuroma are generally obscure. Multiple neuromata are sometimes hereditary, and are probably due, in most cases, to a congenital tendency of tissue growth. Plexiform and multiple neuromata have been met with in the same family. They are said to be sometimes the result of general neurotic predisposition, and evidence of this is found in the occasional occurrence of neuromata in the subjects of cretinism or idiocy, of which some remarkable examples have been recorded. It is now generally held that multiple and plexiform neuromata, together with the affection known as elephantiasis neuromatodes, are results of the same congenital process.[‡] Multiple neuromata are said to be almost confined to men. The isolated form is not uncommon in women. They may occur at any age. Virchow believes that they are unusually common in the phthisical and scrofulous.

Of traceable immediate causes the most frequent is traumatic injury. Pressure, punctured wounds, or division of the nerves may give rise to them, apparently by a perversion in the process by which cicatrisation takes place. A well-known instance is the formation of the so-called "amputation neuromata," or "bulbous nerves," round or oval growths, the size of a bean or larger, which form on the divided extremities of the nerves in the stump left after an amputation.

The occurrence of new growths containing nerve-fibres may seem to be very remarkable. We must remember, however, how frequently they are connected with traumatic causes, *i. e.* take origin in cicatricial processes. In such processes the nerve-fibres present a remarkable power of growth. Ranvier has shown that from the end of each old fibre several new fibres grow, only one of which probably ultimately persists. Under apparently mechanical influences some of these fibres may twist about, and even turn and grow upwards. It is thus not difficult to understand that a true neuroma may result from injury to a nerve; it is said, indeed, that the ends of the nerves in amputation stumps are generally enlarged into minute bulbs, which contain more fibres than do the nerves on which they are situated.*

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[*] 'Med. Times and Gaz.,' 1883, i, p. 152.

[†] Garré, 'Beiträge z. klin. Chir.,' ix; Scheven, ibid., xvii.

[‡] See especially Finotti, 'Virch. Arch.,' cxliii. Some go so far as to include molluscum fibrosum in the same category (Soldau, 'Inaug. Diss.,' Berlin, quoted in 'Virch. Jahrb.,' xxx, i, p. 231).

SYMPTOMS.—These may be entirely absent, especially in the case of multiple (true) neuromata, which are sometimes discovered post mortem when their existence has not been suspected. Often, however, they cause marked symptoms. The most frequent is pain seated in the tumour (which may be extremely tender) or referred to the distribution of the nerve, often acute, burning, or darting, and frequently intermittent. It may be increased by pressure on the tumour. In the case of the terminal neuromata local pain may be the only sensory symptom; but when the nerve-fibres suffer, paræsthesiæ, numbness, or formication may be complained of, and ultimately sensation may be lessened or lost. Paralysis of the muscles supplied by the nerve is occasionally met with, the region paralysed corresponding to the distribution of the nerve on which the tumour is situated, except in the case of neuromata in the cauda equina, from which paraplegia may result, with flexor spasm. A neuroma of the pneumogastric may cause grave cardiac disturbance, sometimes increased by pressure on the tumour.

More common motor symptoms are reflex spasms in adjacent or even distant muscles. For instance, in a case of "bulbous nerves" in the stump after amputation at the shoulder-joint, the muscles on that side of the neck were in constant clonic spasm. Occasionally epileptiform convulsions are produced, and have been known to cease when a painful neuroma had been excised. The occurrence of these symptoms has little relation to the size of the tumour.

When the neuromata are in accessible situations they may be felt. Sometimes they give rise to visible tumours, and in multiple neuroma the course of the superficial nerves may be marked out by lines of bead-like swellings, as in Fig. 47. The cutaneous neuromata also cause visible swellings. The whole abdomen was covered with such nodules in the case recorded by Smith (Fig. 46). They are sometimes not sensitive, sometimes exquisitely tender and most painful, especially when closely united with the skin. Pressure on the nerve above the tumour commonly lessens the pain.

Plexiform neuroma rarely causes other symptoms than the signs manifesting its local presence. The functions of the nerve-fibres involved are not usually interfered with. Once or twice anæsthesia of the skin has been observed.

Course.—The growth of neuromata varies greatly in rapidity. When symptoms occur they most commonly last for a long time, increasing in severity, and from the long-continued pain the patient may ultimately be worn out. In other cases the symptoms show little tendency to increase, and in rare instances they may lessen in severity and even disappear.

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* Bowlby, loc. cit., p. 20.

Diagnosis.—The recognition of the existence of a neuroma depends on its superficial position, or on the production of symptoms of damage to the nerve. The latter, when existing alone, are equivocal, since they may be due to pressure on the nerve from an adjacent growth, or even to neuritis. The disease may be suspected if symptoms—pain, numbness, weakness—occur, limited to the distribution of a nerve-trunk, and no cause of external pressure can be discovered, and the long course of the symptoms makes it unlikely that they are due to neuritis. The diagnosis can, however, only be certainly made when the tumour can be felt. Secondary subcutaneous sarcomata may very closely resemble neuromata.

The diagnosis of the kind of neuroma, whether "true" or "false," *i.e.* composed of nerve-fibres or other tissue, is very difficult. Multiple neuromata are usually true, and of very slow growth; false neuromata are rarely multiple unless they are of infecting nature and rapid in development. The existence of idiocy, or other neuropathic indication, is in favour of the neural nature of the tumours. The lateral position of the growth on the nerve is in favour of its "false" character. The diagnosis of plexiform neuroma is only possible when the tortuous nodular cords can be felt.

Prognosis.—This depends on the existence of symptoms. If these are absent, unless the tumour is of rapid growth, there is good reason to hope that they will not occur. In multiple (true) neuromata the nerve is less likely to suffer than in isolated growths. If symptoms are present the prognosis is less favorable, and depends on the tendency which they exhibit to increase.

Treatment.—Medicines are useless except in the syphilitic forms of nerve-tumour, not here considered. Extirpation is the only remedy. If the tumour is lateral, and can be shelled out, the removal entails little risk. If, however, the growth infiltrates the substance of the nerve, the affected part must be excised, and the ends brought together. The risk of permanent loss of function in the nerve is great, and in deciding on such an operation, the urgency of the symptoms, and the importance of the function of the nerve, must be taken into consideration. After the extirpation of such tumours, the tendency of cicatricial processes in nerves to go on to the formation of similar growths (seen in "bulbous nerves") renders the chance of relapse considerable. The operation reproduces conditions favorable for the activity of whatever predisposing influence aided in the original development of the disease. When neuromata are multiple, surgical treatment is out of the question, except for isolated tumours that cause distress.

In the case of terminal neuromata—whether the cutaneous "tubercula dolorosa" or the amputation neuromata—excision is practicable, and it is unattended with the risks attending interference with a feeble. At the same time this is probably chiefly because too much and too constant work is thrown on the upper thoracic muscles of extraordinary breathing. Any lung disease, such as an attack of bronchitis, is rendered far more serious by the diminished breathing power. When the diaphragm is paralysed, the movement of the thorax is often increased, and the expansion of the lower part may draw forwards the adjacent abdominal wall. This must not be mistaken for the effect of the descent of the diaphragm. Paralysis has to be distinguished from (1) abnormal nervous breathing. The diaphragm is used little in extraordinary breathing, which is chiefly by the upper part of the thorax, of the "superior costal type," as it is called. Hysterical and nervous patients will often breathe, for a time, only in this manner, even when at perfect rest, especially when they are under observation. Such breathing is no doubt facilitated in women by the fact that the diaphragm is habitually used by them less than by men. Repeated examination may be necessary to determine whether there is any real paralysis of the diaphragm in these cases. A single diaphragmatic inspiration settles the point. The patient's attention should be distracted, and she should not be aware of the object of the examination. (2) Inflammation of the diaphragm may arrest its movement, and so also may diaphragmatic pleurisy or peritonitis—each distinguished by the fact that it is usually secondary to adjacent inflammation, and any movement causes characteristic pain. (3) A primary and isolated degeneration of the muscular fibres of the diaphragm has been described by Callender and others as common after death, but it has not yet been proved to cause symptoms, and needs further investigation.

Paralysis may be due to disease of the nerve, its roots, or of the spinal cord. In the latter case other muscles always suffer; in the former the diaphragm usually suffers alone, the result of peripheral neuritis. The distribution and the course of the other palsies are distinctive. By far the most common cause is disease of the spinal cord, local or ascending from below and adding this final effect to the preceding palsy of the intercostals. In disease of the nerve-roots there are other indications of the position of the disease. The affection is often overlooked because not searched for, especially in local disease of the cervical cord. The observer forgets that its associations are not with the lower dorsal muscles, and he omits to look for it in cases in which the cervical cord is suffering and other respiratory muscles act well.

If there is reason to suspect partial neuritis as from cold, hot fomentations at first, and afterwards counter-irritation, should be applied over the lower and inner part of the anterior triangle of the neck. The only other special point in treatment is the application of electricity. The nerve may be stimulated by pressing the rheophore deeply outside the lower part of the clavicular portion of the sterno-mastoid. The other pole may be placed at the epigastrium or over the nerve in its continuity. In amputation flaps the chance of a relapse is considerable.

In plexiform neuroma excision is undesirable, and no other treatment is of any use.

Pain, and the reflex spasm, that are so distressing in amputation neuromata, need relief when attempts to cure have failed. By nerve-stretching we may perhaps give relief without the effects that follow excision, and in cocaine we have a means of arresting for a time the ingoing impulses that play an important part in generating the symptoms. The rest thus given to the centre is absolute, and its degree and frequent repetition may compensate for its brevity.

DISEASES OF SPECIAL NERVES.

The diseases of the cranial nerves may be most conveniently considered in connection with the diseases of the brain. Here, therefore, only the affections of the spinal nerves will be described, in so far as they present special features. The effect of paralysis of the individual muscles has been already described in detail, and need not be here repeated, except in general terms, or in so far as the association of palsies resulting from a nerve-lesion presents a particular character.

Phrenic Nerve.—Impaired function of the phrenic nerve is commonly the result of disease of the spinal cord, or of the roots of the nerve—the third and fourth cervical trunks—from disease of the membranes or the bones. The deep position of the nerve-trunk protects it from injury, but it is occasionally damaged in wounds of the neck, and in its course through the thorax it may be compressed by tumours, aneurisms, &c., or involved in inflammations of the pleura. Paralysis of one nerve sometimes follows exposure to cold, and is ascribed to neuritis. That of both has been met with in multiple neuritis; it has also been observed following diphtheria and influenza, in the course of tabes (Gerhardt) and progressive muscular atrophy, and in poisoning by lead and alcohol. In disease of the spinal cord and membranes both nerves are usually paralysed; causes acting on the nerve-trunk usually affect one only. The effect and symptom of paralysis is inaction of the diaphragm (see p. 33). If one nerve only is affected, the diaphragm does not descend on that side, but the movement of the other side lessens the resulting defect of movement, and it can then be detected only by close observation. The loss of the action of the diaphragm has little effect on the respiratory functions while the patient is at rest, but dyspnœa is said to be readily produced by exertion; the breathing then becomes quick and the voice feeble. At the same time this is probably chiefly because too much and too constant work is thrown on the upper thoracic muscles of extraordinary breathing. Any lung disease, such as an attack of bronchitis, is rendered far more serious by the diminished breathing power. When the diaphragm is paralysed, the movement of the thorax is often increased, and the expansion of the lower part may draw forwards the adjacent abdominal wall. This must not be mistaken for the effect of the descent of the diaphragm. Paralysis has to be distinguished from (1) abnormal nervous breathing. The diaphragm is used little in extraordinary breathing, which is chiefly by the upper part of the thorax, of the "superior costal type," as it is called. Hysterical and nervous patients will often breathe, for a time, only in this manner, even when at perfect rest, especially when they are under observation. Such breathing is no doubt facilitated in women by the fact that the diaphragm is habitually used by them less than by men. Repeated examination may be necessary to determine whether there is any real paralysis of the diaphragm in these cases. A single diaphragmatic inspiration settles the point. The patient's attention should be distracted, and she should not be aware of the object of the examination. (2) Inflammation of the diaphragm may arrest its movement, and so also may diaphragmatic pleurisy or peritonitis—each distinguished by the fact that it is usually secondary to adjacent inflammation, and any movement causes characteristic pain. (3) A primary and isolated degeneration of the muscular fibres of the diaphragm has been described by Callender and others as common after death, but it has not yet been proved to cause symptoms, and needs further investigation.

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NERVES OF THE UPPER LIMB.

The nerves of the arm and shoulder are derived from the five lower cervical and the first dorsal nerves. These interlace in the brachial plexus in such a complex manner that most of the nerves of the arm are derived from many spinal roots.

The nerve-roots form, by their union, three trunks, which we may indicate by Roman numerals. They are formed thus:—I, by the branch from the fourth, and the roots of the fifth and sixth cervical; II, by the seventh; and III, by the eighth cervical and the first dorsal. Each trunk divides into two parts, and the union of these divisions forms the three cords of the brachial plexus from which the nerves of the arm proceed. But before the primary trunks divide, certain nerves arise, the origin of which is thus less doubtful. The fifth and sixth cervical roots give origin directly to the posterior thoracic nerve for the serratus, and from the cord formed by their union springs the supra-scapular nerve. The three cords of the plexus have the following relations:—The posterior is derived from all three primary trunks, and gives rise to the subscapular nerve, the circumflex, and the musculo-spiral (or radial, as the whole nerve is sometimes termed). The upper or outer cord is derived from the two upper primary trunks, *i. e.* from the fourth, fifth, sixth, and seventh cervical roots, and from it proceed one anterior thoracic and the musculo-cutaneous nerves, together with the outer head of the median. The inner or lower cord is derived only from the lowest primary trunk, *i.* . from the last cervical and first dorsal, and gives rise to the ulnar, the inner head of the median, the internal cutaneous, the intercosto-humeral, and to the second anterior thoracic nerves. It may be convenient to put these coarse anatomical relations in the form of a table.

Nerves.		*Primary Trunks.*		*Nerves.*
Subscapular Circumflex Musculo-spiral	Post. cord	I. 4, 5, and 6 C.	Upper cord	External ant. thoracic. Musculo-cutaneous. Outer head of } median.
		II. 6 and 7 C.		
		III. 8 C. 1 D.	Lower cord	Inner head of Ulnar. Internal cutaneous. Intercosto-humeral. Internal ant. thoracic.

These anatomical facts, however, give us little help in tracing the relation of the nerves to the spinal roots. The investigations of Ferrier and Yeo,* who ascertained the movements produced by faradising the several spinal roots in the monkey, show us the way in which the movements, muscles, and nerves are represented in the spinal roots. Their results are therefore of great interest, although we are not justified in transferring the facts to man except in so far as they receive confirmation from human anatomy and pathology.* Subject to this reservation, the chief results are important, and may be thus stated: †

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* 'Proc. Roy. Soc.,' March 21st, 1881. Careful dissections by Herringham (cf. March 25th, 1887) give somewhat different results, but this method of investigation is open to more uncertainties than that of stimulation. Herringham's conclusions should, however, be noted by future investigators.

The roots to which the several nerves are thus traced are as follows:—Subscapular, 6 and 7 C.; circumflex, 4 and 5 C.; musculo-spiral, 4, 5, 6, 7, 8 C.; musculo-cutaneous, 4, 5 C.; median, 5, 6, 7, 8 C.; ulnar, 8 C., 1 D.

Still more important are their observations on the relation of various movements of the arm to the nerve-roots, and these are as follows:

Cervical 4.—Elevation and retraction of the arm, flexion and supination of the forearm; by the rhomboids, supra- and infra-spinatus, biceps, brachialis, and supinators.

Cervical 5.—Similar to the last, but without retraction of the arm, and with extension of the wrist and first phalanges; by the deltoid, serratus, flexors of elbow, extensors of wrist, and long extensors of fingers.

Cervical 6.—Adduction and retraction of the upper arm, extension and pronation of the forearm, flexion of the wrist; by contraction of the pectoralis, latissimus dorsi, triceps, flexors of wrist, pronators.

Cervical 7.—Adduction and rotation inwards at the shoulder-joint, flexion of the wrist, and of the fingers at the second phalanx; by the teres major, latissimus dorsi, subscapularis, triceps, and long flexors of the fingers.

Cervical 8.—Flexion of fingers and thumb so as to close the fist; flexion of wrist towards the ulnar side, pronation of forearm, extension of elbow; by the intrinsic muscles of the hand, the long flexors of the fingers and thumb, the flexors of the wrist and the triceps.

Dorsal 1.—Adduction of the thumb, flexion of the fingers at the metacarpo-phalangeal joints; by the interossei, &c.

Thus most movements are related to many spinal roots. The nerves supplying each muscle have a correspondingly multiple relation to the spinal roots. It cannot be traced anatomically, but has been ascertained by comparing the results of experiments on animals with those of disease and injury in man, and (in the case of sensation) of the division of roots for the treatment of some forms of disease. The facts thus ascertained have been confirmed by cases of disease of the spinal cord itself, which, when sufficiently limited to be significant, have been found to agree closely with observations on the roots. The constituents of each nerve-root come from a definite part of the cord, which is termed the corresponding spinal "segment." A single muscle is seldom related to a single segment; usually its nerve-fibres come from parts of two segments, or even from three. Its representation in the grey matter of the cord corresponds to its relation to the nerve-roots, and has often a considerable vertical extent. The most important relations are these: that of the deltoid, rhomboids, supra- and infra-spinatus, flexors of the elbow, and supinators to the fourth and fifth cervical; that of the adductors of the arm and extensors of the elbow to the sixth and seventh nerves; pronation to the sixth and eighth; extension of the wrist to the fifth; flexion to the eighth; extension of the first phalanx to the fifth; flexion of the fingers to the seventh and eighth, and the action of the intrinsic muscles of the hand to the first dorsal. These facts will probably, in the future, find important practical applications.

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* In the case of the leg there are some important discrepancies between the results in the ape and the conditions that obtain in man. These will be noticed in their proper place.

† Ferrier has since stated that the relations he gave (followed in the text) were all one nerve too high ('Proc. Roy. Soc.,' 1883, vol. xxxv, p. 229), but this would make the innervation of the intrinsic muscles of the hand from the second dorsal nerve, which is certainly not the case in man.

Regarding the relation of the sensory branches to the nerve-roots, we have, besides the facts of anatomy, only the evidence furnished by rare cases of injury or disease in which the lesion and its effects are well defined.* Few facts of this character are, however, so conclusive as that illustrated in Fig. 51, in which the area shaded by slanting lines was rendered anæsthetic by the division of the posterior roots of the last cervical and first dorsal nerves. The general indications are that the distribution of the sensory fibres of the nerve-roots is along the axis of the limb, and not across it. The last two pairs of nerves supply the ulnar side of the limb and the finger tips, the fifth pair the radial side and the middle roots of the brachial plexus, the middle zone of each side of the limb and the thumb.†

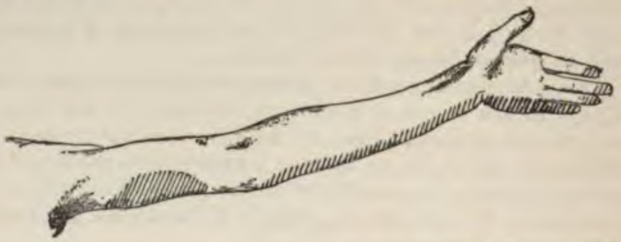

FIG. 51.—Area of impaired sensation after division of the posterior branches of the 8th C. and 1 D. nerves, by Professor Horsley for inveterate paroxysmal neuralgia in that region.

Paralysis of the nerves of the upper limb may be due to disease of the nerve-roots, of the plexus, or of the trunks that arise from these. It is convenient to consider first the diseases of the nerve-trunks, and afterwards those of the nerve-roots and of the plexus. From the roots themselves only one important nerve-trunk is derived:—the posterior thoracic nerve. The others spring from the plexus.

The morbid influences that affect the nerves of the arm are very varied. The brachial plexus passes close to the shoulder-joint, and hence dislocation often damages the nerves, sometimes one only, sometimes several, and occasionally all the trunks. In fractures of the bones the nerves adjacent may be torn, or may be compressed by the ends of the bone, or by the callus that is formed, especially by the exterior "fibrous callus." The musculo-spiral nerve is most prone to suffer from its proximity to the humerus, and in one such case the nerve, when exposed, was found not only to be embedded in the callus tissue, but to have been divided for about one-third of its diameter, opposite the fracture. Bandages that are applied are occasional sources of compression, and there is especial danger of harm from those that secure a splint applied to prevent motion in cases of neuritis. Unless loss of sensation draws attention to it, the injury is generally only discovered when the splint is given up, and therefore its possibility should be remembered. The course of certain nerves is, in some parts of the arm, especially apt to be directly involved in wounds of various kinds, or they may be compressed by the cicatricial fibrous tissue when the wound is merely near the nerve.

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* Instructive facts of this character will be found in papers by Thorburn, 'Brain,' January, 1887, and October, 1887, Allen Starr ('American Journal of the Medical Sciences,' 1892), and Head ('Brain,' 1893 and 1894), who has mapped out the areas representing the segmental distribution of the sensory fibres of the spinal cord by means of observations on superficial tenderness accompanying visceral disease, on cases of herpes zoster, and on cases of spinal cord disease. (See diagrams at end of Chapter on "Functions.")

† This is due to the mode of growth of the arm-bud in the embryo, as shown by Ross in a most instructive paper, 'Brain,' January 7th, 1888. See also chapter on the Functions of the Spinal Cord.

The position of some exposes them to pressure against the unyielding bone. Even slight pressure on these nerves is effective when prolonged, as it often is during sleep, when warning sensations are unperceived. The sleep induced by alcohol is especially apt to be attended by this accident. Hence there is a group of "*Sleep-palsies*" of the arm, chiefly of the musculo-spiral and median nerves, rarely of the plexus itself. One nerve, the musculo-spiral, passing between the bone and the triceps, is occasionally bruised by the contraction of the muscle.

Primary neuritis may involve any nerve, or the brachial plexus, or the nerve-roots; certain nerves are also particularly prone to suffer in peripheral neuritis. The inflammation of the plexus, which is a unilateral perineuritis, is separately described (Brachial Neuritis); the latter is considered in the general account of Multiple Neuritis. Secondary neuritis, set up by injury or by adjacent inflammation, may extend upwards, and, reaching the plexus, spread to other nerves. Neuromata are rare. Various morbid processes in the neck may involve the nerve-roots.

Posterior Thoracic Nerve—Nerve to the Serratus.—In consequence of its position and long course, the nerve to the serratus often suffers. After being formed in the substance of the scalenus medius muscle, by the union of branches of the fifth and sixth cervical nerves, it passes behind the brachial plexus and along the side of the chest to the lower border of its muscle. It is in the neck that the nerve is most often injured; injury to the axilla less often implicates it. It may be damaged in the neck by direct pressure—as in carrying on the shoulder a heavy sharp-cornered object—or by severe muscular efforts, such as carrying a weight that does not press into the neck, wielding a heavy hammer, or long-continued exertion with the raised arm, as, for instance, in whitewashing a ceiling. In such cases a traumatic neuritis is set up by the violent compression of the nerve consequent on the forcible or prolonged contraction of the muscle through which it passes. Whe the onset of the palsy occurs immediately on the exertion, a paralysing injury to the nerve must have occurred, which will be followed by secondary neuritis. Falls and blows on the neck and shoulder are occasional causes; there is usually much bruising of the adjacent parts. Punctured and gun-shot wounds occasionally involve the nerve, which may also be affected in or after typhoid fever, influenza, and diphtheria. The least frequent cause is exposure to cold, such as a draught on the neck, or sleeping on damp earth. I have once known it to follow parturition, developing four days afterwards, doubtless from neuritis excited either by the muscular exertion or by exposure of the neck to cold during the labour. An hysterical paralysis in the distribution of the nerve has been described by Verhoogen. The serratus suffers also in central disease, chiefly in progressive muscular atrophy and dystrophy and infantile paralysis; but in these it is always associated with palsy of other muscles. Isolated paralysis of these nerves is nine times as frequent in men as in women. It occurs chiefly in muscular workers, and during the active period of life, twenty-five to forty. The immediate causes sufficiently explain this relation to age and sex, and they also account for another fact—that it is far more frequent on the right side than on the left. It is sometimes bilateral, but the two nerves are never damaged at the same time. One man was affected on the right side after carrying heavy beams on the shoulder; he then carried them on the left shoulder, and the left serratus became paralysed.

Severe neuralgic pains in the neck and about the shoulder commonly precede and accompany the onset of the affection when due to neuritis. The symptoms of the paralysis of the serratus have been already described (p. 35). It is readily recognised by the recession of the posterior edge of the scapula from the thorax when the arm is put forwards (Fig. 8, p. 36). In severe cases the muscle is found to have lost faradic irritability, although it may still contract to the voltaic current.

The course of a severe case is always tedious. Months may pass before improvement is manifested, and the paralysis is sometimes permanent. In treatment it is desirable to maintain the nutrition of the muscle, as far as possible, by electrical stimulation, and its superficial situation renders this easy. If counter-irritation is desirable, it should be applied over the position of the scalenus, since the lesion of the nerve is commonly at this place. The use of the arm should be limited. When practicable, the elbow should be carried in a sling of such a length as slightly to raise the shoulder. All movements that may involve contraction of the scalenus, *i. e.* all movements needing elevation of the shoulder, must be prohibited.

The *supra-scapular nerve* arises from the trunk formed by the union of the sixth, fifth, and a branch of the fourth cervical nerves, but its fibres come from the fifth and partly from the fourth. It is occasionally damaged, alone or with the circumflex, in dislocation of the humerus, and by falls on the shoulder, and by carrying heavy weights. In some cases the injury may be to the upper part of the plexus (see p. 111), which may recover except this nerve It suffered with the circumflex, in a case recorded by Remak, from acute limited inflammation of the plexus, due to exposure to cold after over-use of the arms. Bernhardt has seen it following a fall upon the hand. The result of its disease is palsy of the supra- and infra-spinatus (p. 37). The latter is the more important, and causes a defect of the rotation outwards of the humerus, interfering with many movements, and, among others, with the movement of the pen along the line in writing. Undue work is thrown on the posterior part of the deltoid and the teres minor; the deltoid may become hypertrophied, and its posterior edge is conspicuous against the wasted infra-spinatus. The scapula is rotated, so that the lower angle is moved upwards and inwards. The paralysis of the supra-spinatus is unimportant, except that the humerus is less firmly fixed and the deltoid more readily fatigued. It can, in very slight degree, supplement the deltoid, and if paralysed with the deltoid, the head of the humerus falls more than it does when the deltoid is paralysed alone. Some anæsthesia over the scapula often results from disease of this nerve; early in the affection there is frequently severe pain in the region of the shoulder-girdle.

The *Circumflex nerve*, although arising from the posterior cord of the plexus, seems to derive its fibres from the same source as the supra-scapular, the fourth and fifth cervical nerves. It supplies the deltoid and teres minor, and the skin over the deltoid. The course of the nerve renders it very liable to injury from dislocations and falls on the shoulder, and from the pressure of a crutch. Simple neuritis and so-called "rheumatic palsy" are rare, but the nerve has been affected in some acute diseases, possibly from pressure, and in rheumatic fever, perhaps by inflammation extending from the joint; typhoid and diabetes may also give rise to circumflex paralysis. It suffers, with other nerves, from disease of the upper part of the brachial plexus, and the chief symptom is paralysis of the deltoid (p. 36), which abolishes almost all power of raising the arm, a very trifling degree of abduction by the supra-spinatus alone remaining. The slight nerve-supply that the fore-part of the deltoid receives from one of the anterior thoracic nerves is insufficient to mitigate the effect of the paralysis of the circumflex, although it may maintain slight power of voluntary contraction in the anterior fibres, and this sometimes gives rise to an erroneous impression that the paralysis of the circumflex nerve is incomplete. The effect of the conjoined palsy of the teres minor is unimportant. The wasting of the deltoid causes a change in the shape of the shoulder (Figs. 7 and 9). Sensation may be lost in the skin over the lower part of the muscle, and the loss sometimes extends on to the shoulder (Fig. 52); Hitzig pointed out many years ago that the anæsthetic area is often the seat of vaso-motor paralysis. In some cases there is no anæsthesia, even when the muscle is wholly paralysed; we have seen that this is often the case in nerve lesions (p. 78). Adhesions are apt to form in the shoulder-joint, in part probably the result of trophic changes, since the circumflex supplies the articulation as well as the chief muscle that moves it.

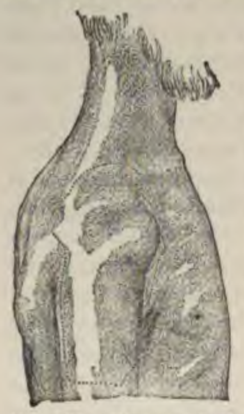

FIG. 52.—Disease of the circumflex nerve from pressure neuritis during acute illness; wasting of deltoid. The dotted line indicates the area of cutaneous anæsthesia.

The diagnosis of the paralysis of the circumflex nerve is easy. The only condition readily mistaken for it is anchylosis of the shoulder-joint in a stout individual, in whom the state of nutrition of the muscle is not readily perceived. The risk of error is increased by the fact that arthritis and paralysis may result from the same cause—a fall on the shoulder. Passive movement at once solves the problem. The scapula moves with the arm in anchylosis, and not in palsy. Furthermore, attempts at voluntary contraction leave the muscle flaccid, while if the affection is in the joint the muscle-fibres become rigid in the attempt to contract. The importance of examining the joint is very great in all cases of interference with movement of the shoulder. A patient had an epileptic fit which was supposed to have left brachial monoplegia, merely because he fell on the shoulder, and the movement of the arm was lessened first by pain, and then by arthritic adhesions. In diagnosing the lesion from those of other nerves, the electrical reactions and the area of anæsthesia may be important.

The *Musculo-cutaneous nerve* supplies the chief flexors of the elbow and the skin over the radial side of the forearm. It is scarcely ever paralysed alone, but often suffers, with other nerves, in disease of the brachial plexus. In the few recorded cases the cause has been traumatic,* but in Remak's case of paralysis of the supra-scapular and circumflex nerves on the right side, from limited neuritis of the plexus, there was complete palsy of this nerve on the left, thought to be due to a still more limited inflammation in the left brachial plexus.† The symptoms correspond to its function; there is paralysis of the biceps and brachialis anticus (see pp. 38, 39), the effect of which is especially conspicuous when the arm is supinated and the supinator longus cannot flex the elbow. There may also be anæsthesia of the radial side of the forearm.

The *Musculo-spiral nerve* is more frequently paralysed alone than any other nerve of the arm. Arising from the posterior cord of the brachial plexus, it seems to derive its motor fibres from all the nerve-roots that enter the plexus except the first dorsal. It supplies the triceps, all the muscles on the back of the forearm, the extensors of the wrist and fingers, both the supinators, also the skin on the radial side of the back of the hand, the back of the thumb, index finger, and half the middle finger. It is thus the extensor nerve of the arm, and has a more complete relation to a single function than is common among the nerves of the limbs; a fact that has given rise to diagnostic error, since a palsy limited to a single function suggests a central cause. The frequency with which this nerve suffers is due to its course. As it leaves the brachial plexus to wind round the bone, its position exposes it to injury in dislocation or from the pressure of a crutch; the most common form of "crutch palsy" is paralysis of this nerve. Lying as it does close to the humerus, it is readily torn in fracture, or compressed by callus, and it suffers gravely from even temporary pressure against the hard bone. Such pressure is often exerted during sleep, especially that induced by alcohol. The nerve may be paralysed by the pressure to which it is exposed when the body, with the arm beneath it, rests on the ground or on a hard bed. Thus a man slept all night on a bench, lying on his right side with his arm beneath him, and woke in the morning with this nerve paralysed. Curiously, a precisely similar event had occurred to him three years before. In other cases the pressure on the nerve is that of a hard and sharp object over which the arm is placed during sleep, as the edge of a chair or the side of a couch. The Continental custom of tying together, behind the body, the arms of a prisoner, often causes paralysis of this nerve, sometimes on both sides. In Russia it is not uncommon in infants, from the popular practice of binding the arms to the body and then laying the child to sleep on its side. I have three times met with paralysis from a violent contraction of the triceps, once during the act of pulling on a tight pair of boots, once from throwing a stone with energy, and once from grasping a lamp-post to avoid a fall during a severe attack of giddiness. In each the nerve was at once completely paralysed; and in the second, in which the palsy was severe, a bruised appearance was observed over the lower part of the triceps. Neuritis due to cold is often assumed to be a cause, and is possibly sometimes effective, but the exposure has usually been during alcoholic sleep, and it is probable that the paralysis is due to compression rather than to cold. The same influence may also have been operative in cases in which the nerve has been paralysed during an acute disease. Although in a case of paralysis during typhus, described by Bernhardt, neuritis was found after death, this was at the spot at which pressure is usually effective, and the inflammation was probably thus excited. Pressure always sets up neuritis, and the diagnosis of a primary neuritis is never justified if the spot affected is one liable to compression. Paralysis of this nerve, with others, has followed the use of an Esmarch's bandage; and a partial palsy sometimes results from the habitual or even occasional injection of ether beneath the skin of the posterior part of the arm.

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* See Bernhardt, 'Erkrank. peripher. Nerven.,' 1897, p. 329.

† 'Neur. Centralbl.,' 1896, p. 578.

The symptoms of disease of the musculo-spiral nerve are paralysis of the extensors of the elbow and of the wrist, the long extensors of the fingers and thumb, and the supinators (see pp. 37—40). All these are paralysed by a complete lesion of the nerve near the brachial plexus. When the lesion is near the middle of the humerus, as it is in most cases of compression, the triceps generally escapes, but not always. The supinator longus is usually paralysed, but escapes if the lesion is below the origin of its branch, and may also escape if the injury to the nerve is incomplete. In sleep palsy it usually suffers, but I have seen it unaffected, in a case in which the ext. carpi radialis was also but little affected, and Bernhardt has noted the escape of the supinator in an otherwise complete paralysis from dislocation of the humerus. The extensor palsy causes characteristic wrist-drop, and loss of the power of extending the first phalanges of the fingers and the thumb (see p. 40). There is sometimes a gradation of palsy from the first finger, in which it is least, to the fourth, in which it is greatest (Fig. 53). The same gradation is seen in lead palsy, and other forms of peripheral neuritis. It is not easy to explain. The action of the flexors is feeble, from the loss of antergic support; in most cases of complete palsy I have found the power of flexion reduced from this cause to one third of the normal. The loss of the power of supination is a grave inconvenience. If an object is grasped firmly the arm becomes pronated. The patient tries to compensate for the loss by putting the elbow against the side and rotating the humerus. The pronators may ultimately become shortened. The over-flexion of the carpus, and its deficient support by the extensor tendons, lead to a prominence of synovial sacs, and perhaps of the bones, at the back of the carpus (Fig. 54). In severe cases the muscles waste, and the maximum circumference of

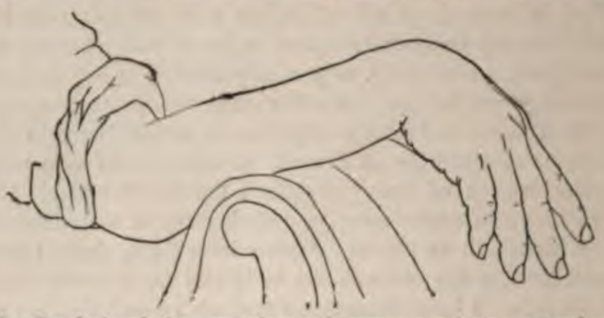

FIG. 53.—Paralysis of the musculo-spiral nerve; maximum extension of wrist and fingers. The extension of the fingers progressively diminishes from the first to the fourth. (From a photograph.)

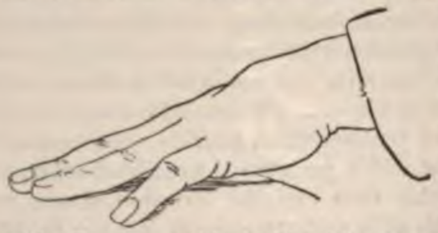

FIG. 54.—Prominence at back of hand from paralysis of the extensors. The patient was suffering from wrist-drop due to silver poisoning (see vol. ii, "Argyria").

the limb below the elbow may be a quarter or half an inch less than on the other side; at the same time there may be thickening of the tendon-sheaths at the back of the hand. The electrical reaction depends on the severity of the lesion of the nerve; commonly there is well-marked degenerative reaction. The affection of sensibility in the area supplied by the nerve is very variable. The skin of the upper arm rarely loses feeling; in the hand sensation may be normal although the muscular paralysis is complete. There may be subjective "tingling" in the part although there is no loss of sensibility. The elbow- and wrist-jerks may be lost even when the reaction of degeneration is not present.

The diagnosis is easy in most cases. The affection is distinguished from lead palsy by its common limitation to one arm, by the affection of the supinator, and often by the sudden onset; that from lead is gradual. The latter is further characterised by the early onset of the degenerative reaction in the affected muscles. These characters, and the commonly obtrusive cause, always suffice for the diagnosis. It must be remembered, however, that this nerve is one most frequently affected in many other forms of multiple neuritis (*q. v.*). The prognosis in disease of the musculo-spiral nerve depends on the severity of the lesion, as indicated by the electrical reaction. When there is evidence of nerve degeneration the paralysis usually lasts for some months. Recovery ultimately occurs in almost all cases. The treatment is that for neuritis, already described, but attention should be paid to the posture of the limb, so as to avoid tension on the affected part of the nerve. For this reason, and also because strong contraction of the triceps may further injure the nerve, extension of the elbow should be avoided in cases of lesion of the nerve as it winds round the humerus.

The *Median nerve* supplies the pronators, the radial flexor of the wrist, the palmaris longus, the flexors of the fingers (except the ulnar half of the deep flexor), the muscles that abduct and flex the thumb, and the two radial lumbricales. Its motor fibres seem to have an extensive origin from all the cervical roots that enter the brachial plexus. It subserves sensation on the radial side of the palm, on the front of the thumb, of the first two fingers, and of half the third finger, and also, at least in many persons, on the back of the last phalanx of the index and middle finger; sometimes also on the adjacent part of the back of the ring finger and the back of the last phalanx of the thumb. Isolated palsy of this nerve is not frequent, and generally results from wounds of the forearm, or fractures of the forearm bones, rarely from injuries in the upper arm. The nerve is occasionally the seat of neuritis, which may be induced by compression, especially in those who, in their work, have to hold an object between the hand and upper arm. Occupation paralyses of this nerve have been observed in ironers, locksmiths, joiners, cigarette makers, and once in a dentist. The nerve may also suffer as the result of pressure of the head of the humerus in sleep or narcosis, while in former days it was not infrequently injured by unskilful venesectors. I have known it to be paralysed at the wrist a few hours after a severe sprain of this joint. Webber has recorded a case in which it seemed to be injured by a violent contraction of the pronator teres. When there is complete damage to the nerve, pronation is impossible beyond the mid-position, to which the supinator longus can bring the forearm, and an attempt is made to supplement this by rotating the humerus inwards, and separating the elbow from the side. The wrist can only be flexed with a strong inclination towards the ulnar side. The thumb is in persistent extension and adduction (like the thumb of the ape, Figs. 22 and 23, p. 44), and cannot be opposed to the tips of the fingers. The power of flexing the second phalanges on the first is lost, and also that of flexing the distal phalanx of the first and second fingers, but this phalanx can still be flexed in the third and fourth fingers by the ulnar half of the flexor profundus (see p. 41). Interosseal flexion of the first phalanx is still possible, and the unopposed extensor action of the interossei on the middle and distal phalanges tends to cause a subluxation of the articulations concerned. The wasting of the thenar muscles is usually conspicuous. The resulting condition is very characteristic; but if the damage is at the wrist, flexion of the wrist and distal phalanges remains. The sensory loss is variable; it may be absolute or absent. If there is anæsthesia it is greater on the palmar surface, and will often be found also on the dorsal aspect of the extremities of the first two fingers. Trophic changes of the skin and nails are not uncommon.

The *Ulnar nerve* comes, through the inner cord of the plexus, from the last cervical and first dorsal roots, and its origin from the lowest part of the cervical enlargement gives it an important relation, since it is the first of all the brachial nerves to be affected by disease that ascends from the dorsal to the cervical region of the spinal cord. The nerve supplies the ulnar flexor of the wrist, the ulnar half of the deep flexor of the fingers, the muscles of the little finger, the interossei, some of the lumbricales, the adductor, and inner head of the short flexor of the thumb. Its sensory area is the ulnar side of the hand, back and front, generally more extensive on the back (two fingers and a half) than on the front (one finger and a half). The course of the nerve, superficial behind the elbow and at the wrist, renders it liable to separate injury. It is often implicated in wounds of the forearm, and about the elbow-joint, by dislocations of the shoulder and elbow, enlargement of the elbow bones, and by fractures of the bones of the forearm. It is occasionally the seat of neuritis, and, like the musculo-spiral nerve, it is sometimes paralysed by pressure. At the elbow the projecting bones preserve the nerve from direct pressure; although Duchenne describes paralysis in men who, while at work, rest the bent elbow on a hard substance, this mechanism is certainly rare. It is much more common for the nerve to suffer in long-continued flexion of the elbow, without external pressure; and from this cause the nerve is sometimes paralysed during sleep.* I have three times seen a sleep palsy of the ulnar nerve. The tension may set up neuritis, and is especially effective if the resistance of the tissues to morbid influences is lessened by general ill-health. Many persons must have noticed that if, when out of health, they sleep with the elbow bent, they wake with tingling, and even loss of feeling, in the region supplied by the ulnar nerve, although this may not happen when they are in good health. If the ill-health is profound, as in an acute illness, local neuritis of considerable intensity may be set up. Thus a lady, a few days after childbirth, who was prostrated by a long, exhausting labour, noticed tingling in the side of the hand, and whenever she bent her elbow, a sensation "as if she had knocked her funny-bone." Paralysis of the muscles supplied by the ulnar nerve followed in a few hours, and in a fortnight there was distinct wasting. When I saw her, six months later, the ulnar nerve behind the elbow was distinctly thickened. She had had a similar but more transient attack after a previous confinement. A similar palsy has occurred during the course of typhoid fever, and both nerves have been known to suffer (Bernhardt). In such conditions not only is the tissue health lowered, but warning sensations are often unperceived. Actual cysts of the nerve have been described by Bowlby and zum Busch.

The importance of the interosseal flexion of the fingers for many actions, such as writing, renders palsy of the ulnar nerve peculiarly disabling. Several illustrations of the symptoms have already been given (pp. 42 and 43). In flexion of the wrist the hand deviates towards the radial side, and persistent distortion may take place. Adduction of the thumb is lost, and so are most movements of the little finger. The fingers cannot be flexed at the first or extended at the other joints, but the loss is slighter in the first two fingers than in the others, because their lumbricales are supplied by the median nerve. In time the opponents of the interossei, by their contracture, lead to over-extension of the first phalanges and flexion of the others; the "claw-like hand" is produced (see p. 43), but this is less complete than in cases of progressive muscular atrophy, on account of the escape of the first two lumbricales. When the palsy is imperfect it may be possible to extend the second and third phalanges, if the first can be over-extended and an advantage thus given to the interossei by the lengthening of the course of their tendons, just as in partial paralysis of the long extensors they may be able to extend the digits if the wrist is flexed, but not if it is extended. In lasting damage the hypothenar eminence may disappear, the palm is hollow, the thenar muscles that remain stand out, and the opponens may cause a slight forward rotation of the thumb.

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* H. Braun ('Deutsch. med. Woch.,' 1894) believes that in sleep the nerve is compressed, not at the elbow but at the shoulder, particularly when the arm is abducted and carried backwards, so that the hands are crossed behind the head. A similar effect may result from the arm being held in this position in an operation, *e.g.* the removal of a breast.

The loss of sensation, as in the case of the other nerves, varies much, both according to, and irrespective of, the degree of lesion of the nerve. Subjective sensations are common, with and without anæsthesia, and those who have had neuritis are sometimes unable to bend the elbow for a few minutes without a sensation of tingling in the area supplied.[*]

The diagnosis of disease of the ulnar nerve is easy. A difficulty is occasionally caused by the circumstance already mentioned, that disease at the lowest part of the cervical enlargement may cause symptoms limited to the region of this nerve, but a knowledge of the fact is usually sufficient to prevent an error.[†]

COMBINED PALSIES OF THE NERVES OF THE ARM.—Paralysis in the region of two or more of the nerves of the arm is very common, and results from many causes. It may be due to disease of the spinal cord or of the nerve-roots within the spinal canal, but with these we are not now concerned. The most frequent causes outside the spinal canal are the following:—(1) Morbid processes in the neck, affecting the nerve-roots outside the spine or the upper part of the brachial plexus, especially growths and exostoses. (2) Diseases of the plexus itself, neuritis or injury, dislocations of the shoulder, the stretching by a sudden wrench of the arm,[‡] or actual rupture of the nerves entering the plexus by great local violence.[§] (3) Fractures of the bones. (4) Neuritis, which may ascend a nerve of the arm to the part of the plexus whence it comes, and there spread; or neuritis of the plexus may be primary and of various extent, "neuritis migrans."

The last, ascending neuritis, occurs when an injury of a single nerve is followed by an extension of the motor and sensory symptoms to the regions supplied by other nerves. This can only be explained by an ascending neuritis reaching the junction of the nerves at the brachial plexus, and there spreading, often in what seems a random manner. Thus a lady cut her wrist so as to leave a scar an inch long, over the position and in the direction of the median nerve. The injury caused paralysis and wasting of all the muscles in the hand supplied by this nerve. This palsy was followed by a gradual loss of power, with diminished faradic irritability, in the long extensor of the fingers, the ulnar flexor of the wrist, and also by defect of sensation in the skin supplied by the ulnar nerve. Hence the neuritis must have ascended to the plexus and there have spread along each root of the median nerve. Again, a woman cut her right hand with a broken stone bottle, along the hypothenar eminence. Three weeks afterwards the muscles of the hand supplied by the ulnar nerve gradually became paralysed, with wasting and loss of irritability, and in the area of the skin supplied by the nerve there was first tingling and then diminished sensibility. Burning pain spread up the front of the forearm, and inner side of the upper arm to the axilla; all the forearm muscles lost power, the flexor carpi ulnaris, and the flexor sublimis digitorum in greatest degree; the tingling and lessened sensibility spread to the fingers supplied by the median nerve, and the abductor pollicis wasted. In this case also there must have been an ascending neuritis of the ulnar, spreading to the median at the brachial plexus. The extension is sometimes only in the neighbourhood of the cause. A whitlow on the thumb was followed by extreme wasting, with reaction of degeneration in all the thenar muscles supplied by the median. But a case is on record in which a nodular inflammation commencing in the ulnar nerves apparently spread up to the sympathetic.[*]

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[*] Anæsthesia of the region supplied by the ulnar and external popliteal nerves is a not infrequent symptom of general paralysis of the insane and tabes. In the ulnar it was first described by Biernacki, 'Neurol. Centralbl.,' 1894, in the external popliteal by Sarbó. See Sarbó and v. Déky, 'Pest. Med.-Chir. Presse,' 1897.

[†] Poncet ('Comptes Rendus,' 1888) describes a manual paralysis in glass-blowers (hooked-hand, main en crochet) which closely simulates ulnar palsy, from which it is to be distinguished by the freedom of the thumb and the absence of anæsthesia.

[‡] Bowlby, loc. cit., p. 225.

[§] Rieder ('Munch. med. Wochenschr.,' 1893) describes a paralysis affecting mainly the circumflex and musculo-spiral nerves of the left arm, and occurring especially in bricklayers from the pressure of the hod upon the shoulder.

The brachial plexus is occasionally the seat of a primary neuritis. This is described in a separate section (p. 119), as it is a defined and important disease.

In dislocation of the shoulder the displaced head of the humerus may damage the nerves. In subcoracoid dislocation they are necessarily compressed. The extent of injury varies greatly; only one nerve may suffer, or not one may escape. Every muscle of the arm may be paralysed, from the deltoid downwards. In most cases the injury is severe in degree; there is rapid wasting of the muscles, with the reaction of degeneration, and there is a great tendency to the occurrence of trophic changes in the skin. It was in a case of this character that the forearm and hand were covered with blisters from the application of water that seemed merely warm to a healthy hand (p. 79).

Fracture of the humerus may damage both the musculo-spiral and ulnar nerves, rarely the median. Fracture of the bones of the forearm often injures both the median and ulnar nerves.

Injuries to the neck sometimes cause a partial paralysis of the arm of peculiar distribution, the special characters and significance of which were first made known by Erb.[†] A similar paralysis may come on apart from injury. The muscles affected are the deltoid, often the supra-spinatus and infra-spinatus, the biceps and brachialis anticus, and the supinators. Erb found that there is one spot between the scaleni, corresponding to the sixth cervical nerve, at which electrical stimulation puts all these muscles in action. Hoedemaker, who has described two cases of this palsy, finds the motor point in a line drawn from the sterno-clavicular articulation to the seventh cervical spine, 1·5 centimetres from the edge of the trapezius. The palsy is apparently dependent on disease of the roots of the fifth and sixth cervical nerves, and the fifth, it will be remembered, receives a twig from the fourth.* Büdinger† and Gaupp‡ have recently shown that in forcible elevation of the arm some backward rotation of the clavicle occurs, compressing the fifth and sixth cervical nerves against the first rib. The fact that in this case the nerve-fibres are simply contused and not broken will no doubt account for the frequency of recovery from this form of paralysis. Besides injuries, this group of palsies may result from non-traumatic processes, probably from neuritis, and also from

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[*] Remak, 'Deutsch. Klinik,' 1864, p. 159.

[†] Heidelberg Society, 1874, 'Ziemssen's Handbuch,' 1874, Bd. xii, pt. 2, p. 509; see also Bernhardt, 'Zeitsch. f. kl. Med.,' 1882, Bd. iv, p. 415. A previous description by Duchenne in 1885 had escaped notice.

FIG. 55.

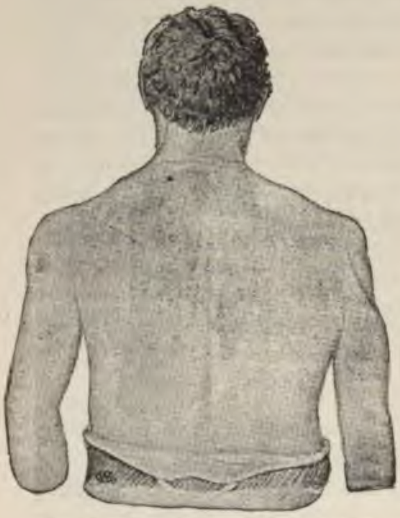
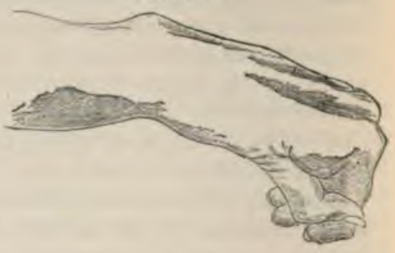


FIG. 56.



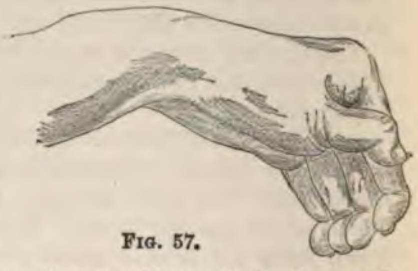


FIG. 57.

FIG. 55.—Combined palsy of deltoid, supra-spinatus, and infra-spinatus, from a fall on the shoulder.

FIGS. 56, 57.—Left hand of a patient suffering from a growth beside the lower cervical spine compressing the nerve-roots. There was anæsthesia of all parts supplied by the brachial and cervical plexus. The arm was adducted, the elbow flexed, the hand in the posture shown, flexion of first, extension of second, strong flexion of last phalanges, the first phalanx of the thumb over-extended, the second flexed. Rigidity extreme, and an attempt to overcome it caused great pain. There was also some contracture of the foot (equino-valgus).

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* Erb referred the palsy to disease of the sixth nerve. The experiments of Ferrier and Yeo point to the fifth and fourth roots as those innervating these muscles, but his subsequent correction indicates the fifth and sixth (see p. 99, footnote).

† 'Arch. f. klin. Chir.,' 1894.

‡ 'Centralblatt f. Chir.,' 1894.

growths in this situation. Duchenne, Seeligmüller, and others have also described paralysis of similar distribution produced in infants during birth, either by pressure from the position of the arm, or by traction on the neck with the finger or hook during turning. Dislocation of the shoulder during delivery may cause very extensive damage to the plexus. Permanent paralysis of each arm was the result of bilateral dislocation caused by a midwife dragging a child out by the two arms.* Most obstetrical cases slowly recover. Those due to injury in adult life are often severe; the symptoms continue for a long time, and may be permanent.

A paralysis complementary to that of Erb, in which the muscles escape which suffer in that form, results from disease of the nerve-roots below.† It is often associated with ocular symptoms, such as narrowing of the pupil and palpebral fissure.

Morbid processes connected with the bones of the cervical spine sometimes produce combined symptoms of irritation and palsy of the nerves of the arm, pain, hyperæsthesia, anæsthesia, paralysis of muscles, and extreme muscular contractures, often very irregular in distribution (such as are shown in Figs. 56, 57). I have several times known sensory symptoms in the arm to be due to a chronic syphilitic cellulitis beside the vertebræ, causing deep-seated induration and severe compression of the nerves; in each case it was completely removed by antisyphilitic treatment. Growths at the part may also compress the subclavian artery, and the weaker pulse may aid the diagnosis.‡

Diagnosis.—The diagnosis of diseases of the nerves of the arm has been for the most part sufficiently considered in connection with the special nerves. It consists chiefly in an application of the facts there stated. One or two more general considerations deserve, however, a brief mention. Some diseases of the spinal cord are first manifested in the arm, by muscular palsy, wasting, or by anæsthesia. The risk of error is prevented, in most cases, by the absence of any correspondence between these symptoms and the functions of special nerves, by the absence of any indication of morbid processes in the neighbourhood of the nerves, and by the presence of other signs of disease of the spinal cord. It has been mentioned that disease in the lowest part of the cervical enlargement may be manifested only in the region of the ulnar nerve, the lowest in origin of all the brachial nerves. Such disease is, however, usually bilateral, and secondary to disease in the dorsal region of the cord, the indications of which have preceded the symptoms in the arm. A knowledge of these facts, an a careful consideration of the distribution and course of the symptoms, will rarely leave the observer in doubt.

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* Danchez, 'Ann. de Gyn.,' &c., Oct., 1891. Double palsy in adults is occasionally seen, as in a case recorded by Bernhardt, in which the arms were pulled on for an hour to steady the patient during the operation of oöphorectomy.

† It has been termed "Klumpke's paralysis," because investigated in Vulpian's laboratory by Mdlle. Klumpke in 1885 (see 'Zeitsch. f. Nervenh.,' 1891); it had, however, been described long previously, notably in 1827 by Flaubert.

‡ Bowlby, loc. cit.

TREATMENT.—There is little in the treatment of the diseases of the nerves of the arm that requires special mention. The chief measure is the treatment of the cause of the paralysis. Any present source of pressure must be removed as far as possible. The ends of a divided nerve must be sutured. It is remarkable how quickly this has sometimes been followed by the restoration of conducting power. The posture of the arm must, in all cases, be such as to avoid tension on an injured or inflamed nerve. The treatment of neuritis is described in the next section. Electrical stimulation of the muscles is of great importance in all cases of severe damage to the nerves. Even in old and stationary cases it sometimes starts some improvement.

NERVES OF THE LOWER LIMB.

Disease of these nerves is far less common in the lower than in the upper limb, with one important exception,—the primary disease of the sciatic nerve that goes by the name of "sciatica." This affection is reserved for separate description.

The nerves of the leg are derived from the lumbar and sacral plexuses. The *lumbar plexus*, consisting of the first three and a half lumbar roots, supplies the skin of the lower part of the abdomen, of the front and sides of the thigh, of the inner side of the lower leg and foot. It supplies also many muscles—the cremaster, those that flex and adduct the hip-joint, and those that extend the knee. Its branches for the leg are the obturator and anterior crural nerves. The *sacral plexus* consists of the fifth lumbar root and half the fourth (lumbo-sacral cord) and the first four sacral nerves, of which, however, only the upper three have to do with the leg. This plexus innervates the extensors and rotators of the hip, the flexors of the knee, and all the muscles that move the foot, together with the skin of the gluteal region, the back of the thigh, the outer side and back of the lower leg, and most of the foot. Its chief nerves are branches to the outward rotators of the hip, the gluteal nerve, and the small and great sciatic.

The results obtained by Ferrier and Yeo (see p. 99) on stimulating the roots of the lumbar and sacral plexuses in the monkey may be thus summarised:

Lumbar I and II. Lower abdominal muscles (not cremaster).

III. Psoas and iliacus, sartorius, extensors of knee; (flexion of hip and extension of leg).

IV. Glutei, adductors, extensor cruris, peroneus longus; (extension of hip and knee, elevation of outer side of foot).

V. Glutei, hamstrings, and all the muscles in front and back of lower leg; (rotation of thigh outwards, flexion and rotation inwards of leg; extension of foot with elevation of outer edge; flexion of distal phalanges of toes).

Sacral I. Hamstrings, calf muscles, long flexor of toes, intrinsic muscles of foot; (slight outward rotation of thigh, flexion of knee, extension of foot, adduction of great toe, flexion of first phalanges of all toes and of both phalanges of great toe).

II. Intrinsic muscles of foot; ("interosseal" flexion of toes, similar to the last).

These results cannot be simply applied to man. For instance, it is certain that in man, one flexor of the hip, the psoas, and the cremaster, are largely innervated from the second lumbar, but no indication of the action of either of these could be observed on stimulating this root in the monkey.

The distribution of the sensory fibres of the nerve-roots is considered in the chapter on the Spinal Cord.

The *Lumbar plexus* itself is sometimes damaged by growths in the abdomen (especially by those that spring from the lumbar glands) and by psoas abscess, while the nerve-roots may suffer in disease of the bones of the vertebræ and in meningitis. The plexus may also be invaded by inflammation ascending the lumbo-sacral cord from the sacral plexus, and it is occasionally the seat of spontaneous neuritis. The *obturator nerve* is rarely affected alone; it has been sometimes damaged in the course of parturition. The *anterior crural nerve* has suffered from the same cause, and is sometimes injured by wounds of the groin or thigh, or by dislocation of the hip-joint. Symptoms may be limited to it in disease of the nerve-roots, and as in injury or growths of the vertebræ, its paralysis occasionally follows acute or subacute inflammation of the knee-joint.

Of the interference with movement that may result, the paralysis of the flexors of the hip depends on the position of the disease. This paralysis is total only when the lumbar plexus is damaged. Disease of the *anterior crural* nerve, within the abdomen, does not affect the psoas, but may paralyse the iliacus, and so weaken, without abolishing, the power of flexion. The chief symptom of disease of this nerve is the loss of power in the extensors of the knee, and the wasting of these muscles, together with loss of the knee-jerk which results from the interruption of the reflex arc. The effect of these palsies on movement is very serious (see pp. 46, 47). Anæsthesia involves the whole of the thigh (except a strip along the middle of the back), the inner side of the leg and foot, and the adjacent side of the first and second toes. In many cases the paralysis is incomplete, and the symptoms present corresponding variation. Irritation of the nerve may cause severe pain in the region supplied by it. This is sometimes an early symptom of a growth near the spine. The pain in neuritis of the plexus may extend along the course of the ilio-hypogastric, ilio-inguinal, and genito-crural nerves, to the lower part of the abdomen and groin.

Impairment of sensation in an area on the front of the lower half of the thigh is occasionally met with as an isolated symptom, usually in men in the second half of life. It comes on without pain, and may pass away after months, or may persist for years. The loss is greatest in the middle of the region, but the transition to normal sensibility is well defined; its origin and nature are mysterious, except that the subjects of it are usually gouty. It seems to have little significance.

Paralysis of the *obturator* nerve causes a loss of the power of adduction of the thigh, so that the patient cannot put one leg across the other. Rotation of the thigh outwards is interfered with, but in slighter degree than adduction. The effect of these palsies (described on p. 47) is far less serious than is the disability which results from disease of the anterior crural. There is disturbance of sensation along the inner side of the thigh and knee; an obturator hernia may press upon the nerve, giving rise to paresis of the muscles which it supplies, and burning pains in the region of its sensory distribution.

The *superior gluteal nerve* occupies an intermediate position between the two plexuses, arising as it does from the lumbo-sacral cord, which descends from the fourth and fifth lumbar roots. Its disease, which is very rare in isolated form, causes paralysis of the gluteus medius and minimus, with a loss of abduction and circumduction of the thigh (see pp. 45, 46).

The *Sacral plexus* is sometimes damaged by growths in the pelvis, by pelvic inflammation of various kinds, and by compression during parturition. In the child it may be damaged by traction on the legs. It is also often the seat of neuritis, which, however, less frequently begins in the plexus than ascends to it from the sciatic nerve. Apart from spontaneous neuritis, which will be separately described, the sciatic nerve outside the pelvis is occasionally injured by wounds, rarely by dislocations of the hip, often by disease of the bone, and by adjacent morbid growths. It is also a relatively frequent seat of neuroma. Of the terminal branches, the external popliteal, by its superficial course, and proximity to the hard bone, suffers from traumatic lesions of various kinds; it is also prone to spontaneous neuritis. This nerve is homologous with the musculo-spiral nerve of the arm, and presents an analogous liability to disease; it occasionally suffers in persons whose occupations entail much kneeling. The posterior tibial nerve is more secure in its deeper course, but may be damaged by fracture of the bones.

The symptoms of palsy of the *sciatic* nerve vary much in their character, according to the position of the disease. The *small sciatic* is implicated only when the mischief involves the pelvic plexus, and it scarcely ever suffers alone. The effect is palsy of the gluteus maximus, which interferes with rising from a seat more than with walking (see p. 45). There is also anæsthesia of the skin in the middle third of the back of the thigh, and in the upper half of the calf. A lesion of the sciatic nerve, near the sciatic notch, paralyses the flexors of the leg (which are also extensors of the hip; see p. 47), and all the muscles below the knees. Often the lesion is below the upper third of the thigh, and then the flexors of the leg escape. Even when all the muscles below the knee are paralysed, walking is still possible, the foot being raised by over-flexion of the hip. The anæsthesia that results from a lesion of the nerve below the origin of the small sciatic, involves the outer half of the leg, the greater part of the dorsum of the foot, and all the sole, but the leg may escape, perhaps through union with other nerves.

The symptoms of disease of the branches are as follows:

That of the *external popliteal* or *peroneal nerve* causes paralysis of the tibialis anticus, long extensor of the toes, peronei, and extensor brevis digitorum. The effect of this is a loss of all power of flexing the ankle and of extending the first phalanx of the toes (see p. 50). The foot cannot be raised from the ground in walking, and talipes equinus ultimately results (Fig. 30, p. 51), which may be attended with persistent flexion of the first phalanges of the toes from contracture of the unopposed interossei. There is also anæsthesia on the outer half of the front of the leg, and on the dorsum of the foot.*

Disease of the *internal popliteal nerve* paralyses the popliteus, calf muscles, tibialis posticus, and long flexors of the toes, as well as the muscles of the sole. In addition to the disability which characterises paralysis of the plantar muscles, there is loss of the inward rotation of the flexed leg if the disease is so high as to involve the branch to the popliteus, and there is also loss of the power of extending the ankle-joint. Talipes calcaneus results (Fig. 28, p. 50). The sensory loss is on the outer lower part of the back of the leg, and on the sole, but varies much.

The *plantar nerves* rarely suffer alone. A lesion of the *internal nerve* causes anæsthesia on the inner part of the sole, and plantar surface of the three inner toes and half the fourth, together with paralysis of the short flexor of the toes, the plantar muscles of the great toe (except the adductor), and of the two inner lumbricales.

Disease of the *external nerve* produces anæsthesia of the skin on the outer half of the sole, the little toe and half the fourth, paralysis of the flexor accessorius, the muscles of the little toe, all the interossei, the two outer lumbricales, and the adductor of the great toe. The effect of this palsy (see p. 52) is serious, since the toes cannot take their proper share in propelling the body forward in walking, and they gradually become flexed at the last two joints and extended at the others, from the contracture of the opponents of the interossei,—a position of the toes that causes serious inconvenience in walking.

DIAGNOSIS.—The diagnosis of diseases of the nerves of the leg is determined by the same general principles as those that have been mentioned as applicable to the nerves in general, and to the nerves of the arm in particular. The limitation of the symptoms to the functional areas of individual nerve-trunks, the evidence afforded by nutrition, irritability, and reflex action that the muscles are separated

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* Anæsthesia in the district supplied by this nerve may be a symptom of various progressive nervous diseases; it was first described in this connection by Sarbó (see p. 110, footnote).

implication of the sensory functions, and
the affected nerves, indicate, in most cases,
To these signs are often added other indica-
corresponding, in its position, with the nerve

to nerve-roots, although by no means
in the case of the nerves of the leg
This is especially the case in the lumbar
occasional difficulty in diagnosis. Pressure
at the level of the origin of the fourth
symptoms identical with those of a partial
nerve. For instance, I have known para-
loss of the knee-jerk, with anæsthesia
the result of a gumma at the side of the
evidence of a spinal lesion is rarely absent
and in this patient a foot-clonus, due to the
left no doubt as to the situation of the
from the long course of the nerve-roots
which may simulate that of the nerves of
region is most commonly due to syphilis
bifida), or new growths.* Bu
bilateral in consequence of the pro
the two sides. In all cases in wh
there is evidence of a disease kn
such as multiple neuritis), the sugge
where the motor or sensory pat
can be affected by a single lesion
the spinal canal. But here, as in
diagnostic rules. Disease of the
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Fr. Schulze, 'Deutsche Zeitsc

does not differ from that of disease of the nerves of the arm. More care, perhaps, is needed to avoid increasing present mischief, or inviting a relapse, by exposure to cold or by fatiguing exertion. More care is also needed to obviate the tendency to secondary contractures in the case of palsies of long duration, and in those attended by pain in which the patient seeks ease in postures to which the muscles only too readily adapt themselves. The contraction of the hamstrings, from constant flexion of the knees, occurs very readily and is most troublesome; that of the calf muscles, which occurs when the flexors of the ankle are paralysed, also constitutes a serious obstacle to walking after recovery. A little timely care, by attention to posture, will often save a vast amount of later trouble. That of the calf muscles, however, which is due to the extension produced by the weight of the foot as the patient lies, cannot always be entirely prevented, but may be lessened by a board or large sand-bag against which the feet can rest.

An outline is given elsewhere of the forms of local inflammation of these nerves, with special reference to the morbid process, on account of its important practical relations. (See Crural Neuritis, p. 133.)

BRACHIAL NEURITIS.

Besides the forms of inflammation of the nerves of the arm already described, a primary inflammation of the brachial plexus occurs in a form as well defined as sciatica, and equally meriting a separate description. This, however, need only include its special features; those that are common to other forms of neuritis have been already described. It is closely analogous to sciatica, and is usually a perineuritis,—a primary inflammation of the sheaths of the branches that enter and form the brachial plexus.

Cases occur, however, in which the symptoms correspond in general character to the rest, but in which their distribution suggests that the nerve-roots rather than the plexus are the seat of the inflammation, and pain about the spine supports the opinion. This form may be called *Radicular Neuritis*. Although its existence has not been established by pathological evidence, the symptoms mentioned admit of no other explanation. A knowledge of their significance is of great practical importance on account of the closeness with which they may simulate those of organic disease of the bones of the spine, or a growth in the spinal membranes.

Causes.—The influence of gout in causing local neuritis is very conspicuous in the brachial form, with the special characteristic that this occurs chiefly late in life, very often from the inherited disease,

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* See Thorburn, 'Brain,' 18
1894, vol. v.

which may be much increased during and after the paroxysms. Loss of sensation is rare, and is met with only in cases that are not only severe but prolonged. The two may concur, as "anæsthesia dolorosa."

The muscles usually present the flabbiness and slight wasting common in neuritis, but the damage to the motor fibres is seldom sufficient to cause considerable atrophy. Sometimes, however, there is enough damage to cause wasting of some group of muscles, with the reaction of degeneration. This is most frequent in the radicular form, in which the damage is to the upper part of the plexus and nerve-roots. There may then be anæsthesia of the skin over the affected muscles. It is difficult to ascertain the existence or the amount of motor weakness; effort induces pain so readily that the patient can seldom be induced to make an attempt to exert force. Power is often said to be almost lost, when it is probably greater than is believed. Besides the muscles, the subcutaneous tissue of the limb may also waste, and the skin may become thin and shining, and present the aspect already described. Subcutaneous œdema is also common, and in one case erythema nodosum was found.* Arthritic changes in the joints of the fingers are almost constant in the cases that occur in later life; the adhesions may be permanent.

Diagnosis.—Few maladies, as a fact of experience, give rise to greater diagnostic difficulty. This is due to several causes: the affection is rare; its symptoms are sometimes equivocal; the subjects are usually in the degenerative period of life, when many obscure diseases attended with pain in the arm occur to the mind of the physician; and lastly, the distinction between neuralgia and neuritis is often difficult, although less so than is supposed. The last is, indeed, the most frequent source of error. The most severe and characteristic cases of brachial neuritis are frequently mistaken for pure neuralgia, on account of the paroxysmal character of the more severe pain, and because the characters of neuritis are unfamiliar. The points to determine the diagnosis are those described in the sections on neuritis and neuralgia;—the degree of persistent tenderness of the nerves and the influence of movement, together with the history of the attack, the locality of the pains, and especially any evidence of damage to the fibres. A history of neuralgia in the person or the family is of slight weight only; the tendencies to true neuralgia and to neuritis are often combined, and each is a frequent result of gout. A far greater difficulty is presented by the cases in which the inflammation remains slight and is confined to the plexus, affecting branches where the conducting fibres can be so irritated as to cause distant pain, which then becomes the leading symptom. This is often paroxysmal, and, when on the left side, may radiate to the chest and be associated with disturbed action of the heart. Angina pectoris is often thought of in such cases; and here again the degenerative age may increase the difficulty by leading to the presence of some coincident disease of the

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* v. Frankl-Hochwart, 'Wien. klin. Wochenschrift,' 1897, No. 1.

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quences are the alterations in the joints. These are very frequent: the fixation occasioned by the pain permits, and the tendency to perverted nutrition produces, adhesion between the articular surfaces, in consequence of which the movement becomes permanently restrained. It is probable, moreover, that the constitutional state which underlies the primary malady often increases the degree of these joint-alterations. The shoulder, wrist, and fingers are the joints that are most frequently thus rendered stiff. The interference with the movement of the fingers is especially troublesome; the joints become painful in consequence of the unavoidable strain on the tissues when an attempt at use is made.

TREATMENT.—The treatment that has been described, as needed in neuritis generally, is suitable also for this form, and its details need not here be repeated. Abstinence from movement is of great importance; the occurrence of stiffness of the joints in consequence of the immobility of the limb must be risked. It is doubtful, indeed, whether the tendency to this is much increased thereby, for the effect of pain, in preventing sufficient movement to maintain the normal state of the joints, is not materially increased by rest otherwise enforced; while the difference between a little movement and none, during the acute stage of the affection, is enough to make a considerable difference in the effect on the inflammation of the nerves. The influence of cocaine, injected under the skin, is very beneficial, but it is needed frequently in severe cases, and two injections (each $\frac{1}{10}$—$\frac{1}{8}$ gr.) may be given daily during the height of the disease. It is important that the nerves should not be irritated by massage in the state of active inflammation; indeed, no therapeutic measure which occasions pain can do as much good as harm. After the tenderness has subsided, gentle rubbing, such as is agreeable, may be employed with advantage. Electricity is only needed for muscles that present the reaction of degeneration, or to aid in removing after-pains. In one severe case, associated with multiple neuromata of the skin, relief resulted from excision of the outer and inner cords of the plexus. The excised portions showed some interstitial neuritis.*

SCIATICA.

The most frequent of all forms of inflammation of a single nerve, unilateral, and primarily of the connective-tissue elements, and especially of the sheath, is that of the sciatic. The fact, coupled with the prominent character of the painful and disabling symptoms, have raised "sciatica" into a conspicuous position among diseases of the nerves, and indeed among diseases generally. This renders desirable a specially full description of the malady, fuller than strictly corresponds with its subordinate position as a variety of neuritis.

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* Maury and Dühring, 'Amer. Journ. of Med. Sci.,' 1874; a similar case of septic origin recorded by Sands and Seguin was not cured by excision of the plexus.

As the word "sciatica" is commonly used, it is a general designation for all affections of which the chief symptom is pain in the region of the sciatic nerve. In a stricter use of the word, however, it is applied to painful affections of the nerve not due to any morbid process outside it; thus limited, it practically corresponds to inflammation of the nerve. Two varieties may be distinguished, however, according as the process in the nerve is excited by primary disease in its vicinity, "secondary" sciatica; and "primary" sciatica, when the pain is the expression of disease beginning in and related to the nerve itself. Primary sciatica is sometimes regarded as a neuralgia. This view is in the main erroneous; the vast majority of cases of sciatica are really cases of true neuritis.

CAUSES.—Sciatica is far more frequent in males than in females; the proportion has been very variously estimated. The best evidence of the incidence of the disease is that presented by the patients treated at the Devonshire Hospital, Buxton, of which an analysis of 1000 cases has been made by Dr. E. V. Gibson.[*] The percentage of males is 88·4, of females 11·6,[†] giving a ratio of 5 to 1, which is probably very near the truth. The disease is unknown in childhood, and rare in the second decade of life. It does not, indeed, occur under fifteen; in more than half the cases it begins between thirty and fifty, and between fifty and seventy in a quarter. Having regard to the smaller number of persons living, the frequency of its occurrence must be regarded as at least as great in late as in middle life.[‡]

No special influence of sex is distinguishable in the decade forty to fifty: the proportion of males to females is that of the whole series. Under forty a smaller, and over fifty a larger proportion of females suffer. Exposure to the exciting causes is probably more common in males during middle life, when also the malady is promoted by the various accelerating influences which aid the active development of its chief constitutional cause—gout. But it must not be forgotten how comparatively small is the actual number of cases in women.

Underlying most cases of sciatica is either the state of definite gout, or that rheumatic diathesis in which the fibrous tissues suffer, especially those that are connected with the muscles, a form closely connected with common gout by co-existence or descent. It occurs frequently in those who are themselves gouty, who present the personal characteristics of the disease, and have fostered it by mode of life. It occurs also in those of gouty inheritance, but who themselves have been abstemious, and sometimes present a weakly constitution—thin, pallid, neurotic. The latter have often suffered from acute articular rheumatism in earlier life. It is among them that the most severe cases are met with, in which the inflammation spreads to other nerves, or involves the sheath of the sciatic with other structures, and that it develops early in life, during, for instance, the second fifteen years. This constitutional state, with all its effects, is sometimes met with when there is only trifling evidence of inheritance. Some unknown influence seems to determine its development in intense degree as a congenital tendency, manifested by rheumatic troubles, various and severe, early in adult life—the sporadic occurrence of that which is usually inherited. There is no evidence of a direct causal relation to other constitutional diseases. Syphilis has been supposed in some cases to give rise to it, but the cases are rare, and the common cause can seldom be excluded.

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[*] Dr. E. Valentine Gibson, 'Lancet,' 1893, vol. i, p. 860.

[†] 884 females, 116 males, in the 1000.

[‡] The percentage distribution of the 1000 cases at Buxton is as follows:—The age is taken at which the first attack commenced. Between 15 and 20, 14 cases; 21 to 30, 159 cases; 31 to 40, 310 cases; 41 to 50, 248 cases (one quarter of the whole); 51 to 60, 187 cases; 61 to 70, 71 cases; 71 to 80, 11 cases.

An exciting cause is to be traced in many of the cases. Exposure to cold is the most frequent. It is usually local exposure, as by wet boots, standing in water, &c.; sometimes, however, a general chill of the body determines an attack. The exposure to cold may be even more direct, as by sitting on wet grass. Draughty water-closet seats are answerable for some cases. The neuritis often arises by the extension of an adjacent rheumatic affection of the fibrous tissue, especially of that form of "lumbago" which involves the fibrous attachments of the muscles at the back of the sacrum, less commonly in its ordinary lumbar seat. This affection passes down from the sacrum, extending along the fasciæ, to the nerve-sheath in the neighbourhood of the sciatic notch; or passes forwards, over the crest, to the front of the iliac bone, and spreads in the tissue about the lumbar plexus, and descends to that which covers the mass of sacral nerves from which the sciatic proceeds. (The fact is of much interest, because it shows that this form of fibrous rheumatism, of which we have only a very vague pathological conception, must be regarded as inflammation, since positive neuritis results from it.) The rheumatic pain has usually existed for a few days only before the extension occurs, but sometimes a chronic affection, after existing as such for several weeks, spreads acutely.

Mechanical disturbance sometimes excites the disease, and often co-operates with other influences. The pressure of the edge of the chair, in those who sit much, is the most frequent. Muscular over-exertion, suddenly compressing the nerve in the thigh, is occasionally effective. If the nerve is already tender, a strong contraction of the muscles at the back of the thigh, especially when the knee is flexed, and the muscles can freely shorten and widen, may produce acute pain in the nerve, evidently by its compression. This cause is probably effective only in a predisposed person, or when there is already commencing neuritis

Various morbid processes within the pelvis may cause sciatica by compressing the sacral plexus, or by exciting inflammation which invades the nerve. Rectal and other tumours give rise to progressive pressure, and the inflammation excited descends the nerve, resembling the primary form. It may be an early symptom of a growth springing from the bone, as an enchondroma arising at the sacro-iliac synchondrosis. Pelvic inflammation and injury during labour are occasional causes. A loaded rectum may be the excitant, but is a rare cause, although apt to be recurrent when once effective. Lastly, the sciatic nerve may be secondarily involved in mischief that is outside the pelvis. The most frequent cause of this is disease of the bone, as disease of the hip-joint, especially senile rheumatic inflammation (Gibson).

The proclivity of the fibrous tissue of this nerve to suffer primarily is due to its position and the exposure this involves, and to its connections, which facilitate the passage of inflammation to it. But there are cases with a strong disposition for fibrous rheumatism to fix itself in the tissues of the pelvis, sacral and lumbar regions; the nerves cannot escape implication; whether the sciatic suffers early or late, and in what degree, depend on secondary conditions, but it is specially liable on account of the anatomical relations of its origin. The mass of the "sacral plexus" is prolonged into it, and the membranes covering this, and its branches, including the lumbo-sacral cord, are very liable to be the seat of such inflammation, wide-spread, fixing itself irregularly on the various nerves, but in special degree on the sciatic. In these cases the pain is extensive, and often severe in the front of the thigh, but the symptoms of descending neuritis are prominent chiefly in the sciatic.

SYMPTOMS.—The two nerves suffer with nearly equal frequency, but Gibson found, in his extensive series, that the left was a little the more liable, in the proportion of 48 to 44 per cent. In about 7 per cent. both were affected simultaneously—a striking indication of the smaller relative part played by the general blood-state, compared with polyneuritis, and the preponderant influence of local excitants. The chief symptom of primary sciatica is pain along the course of the nerve-trunk, often also along that of its branches; pain in the area of its distribution is sometimes subsequently developed. The affection may begin suddenly, especially in cases of rheumatic origin, —as suddenly as lumbago. Some movement seems to excite it, but there has usually been slighter rheumatic pain in the neighbourhood for a day or two, generally about the hip or sacrum. More frequently the onset is gradual; slight pain is felt along the back of the thigh, on movements and in postures that make the nerve tense, or cause pressure upon it. This pain, due to a slight degree of inflammation, has generally existed for some weeks, increasing in degree until a considerable severity is attained, or suddenly becoming intense under the influence of some exposure or over-exertion. At last the patient is easy only when at rest, and when the leg is in a certain posture. Any movement that makes the nerve tense causes pain, and to avoid this the knee, in walking, is kept slightly flexed, and the leg held stiffly so as to avoid stretching the nerve. As the pain on movement increases, spontaneous pain is added, at first chiefly felt in the nerve-trunk, but soon spreading to its branches and distribution. It is usually most intense in certain spots—(1) above the hip-joint, near the posterior iliac spine, (2) at the sciatic notch, (3) about the middle of the thigh, (4) behind the knee, (5) below the head of the fibula, (6) behind the external malleolus, (7) on the back of the foot. The pain may radiate over the whole distribution of the nerve, but it is often so distinctly limited to the course of the trunk and branches, that the patient points these out with exactness when he indicates its course. The chief intensity of the pain is usually down the back of the thigh. It may be dull or acute, is often burning in character, and worse at night. It may seem to dart downwards, starting from the highest point. As the pain on movement increases, the nerve-trunk becomes extremely tender to pressure. Even before the tenderness becomes considerable in the thigh, pain may often be produced in the following manner:—Let the patient sit on a chair with the knee at a little more than a right angle, and the body bent forward, so as to lengthen the course of the nerve at the hip- and knee-joints. If the finger is then pressed into the popliteal space, so as to make the nerve a little more tense, a pain is felt in the course of the nerve at the back of the thigh or above the sciatic notch, and behind the hip. It is due to the sensitiveness of the nerve to tension, and is a very useful test, especially when the part inflamed is high up within the pelvis. It may reveal the affection of the nerve here, by making it more tense, when there is no tenderness to pressure at the back of the thigh.

Abnormal sensations other than pain are often felt in the area of distribution of the nerve,—tingling, formication, and the like; and in severe cases there may be diminished sensibility on the back of the thigh, on the leg, or the foot. The affection of sensibility at the back of the thigh indicates that the disease extends up the nerve, above the sciatic notch, to the origin of the small sciatic, or that this is involved in a simultaneous neuritis. In severe cases the muscles supplied by the nerve become flabby, tender to the touch, and sometimes distinctly weak and wasted. This is chiefly noticeable in the calf muscles, and in the group supplied by the external popliteal nerve. A tendency to cramp in the muscles is often very marked. There may be an alteration in the electrical irritability, usually a slight increase to each form; it is considerable, and amounts to a distinct degenerative reaction, only in very severe cases. Slight fever and corresponding constitutional symptoms may attend an onset that is acute, when the inflammation is intense. Chronic cases, however, are usually not attended by elevation of temperature.

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aiding in the generation of their acute gouty effects on the sciatic. Such a combination may puzzle the observer.

A rare complication is a tendency of the mischief to ascend the nerve. Thus symptoms may spread from the region of the sciatic to that of the lumbar plexus; the pain may spread to the front of the thigh, and the extensors of the knee may become flabby and weak. This is probably due to the passage of an ascending neuritis up the lumbo-sacral cord. In extremely rare cases the morbid process has apparently reached the spinal cord, and indications of cord disease (*e.g.* disordered sensibility in the soles) have thus succeeded those of a primary and apparently simple sciatica. There may even be symptoms of lateral sclerosis (excessive knee-jerk, rectus clonus, &c.), such as develop in some cases of arthritis and other conditions of prolonged pain.

The wider distribution of the pain and other symptoms, when a rheumatic inflammation is extensive from the first, may have the aspect of a complication, but often the involvement of the sciatic does not dominate the case, which is then rather one of general crural neuritis, the result of the affection of the fibrous tissue of the floor of the pelvis where numerous nerves arise. The position of some of them determines the frequency with which they suffer specially, but the size of the sciatic nerve-mass necessarily causes its affection to be chiefly conspicuous. That of some of the lowest branches of the lumbar plexus also determines the common seats of the effects of extension upwards.

In spreading cases, one effect of extension is of particular importance—the paralysis of the bladder that may result from extension to its nerves. The nearer part of the lumbar plexus is often then involved, and pain is felt in the front and outer part of the thigh, and even in the groin and lower part of the abdomen. The vesical nerves from the pelvic plexuses of the two sides are near together, and the palsy of the bladder is often complete, and of long duration, outlasting, it may be, all other symptoms. Fortunately, it is a very rare complication.

PATHOLOGY.—The evidence of pathological changes is scanty. Rheumatic inflammation of the nerve is scarcely ever met with in its active stage; the disease is not one which is even attended by death from other causes, and the morbid changes are a matter of inference. There is, indeed, hardly any organic disease so common as sciatica, of which, in its various degrees, opportunities for pathological observation are so few and so meagre. Signs of inflammation have been found, chiefly conspicuous in the sheath of the nerve, and invading the interstitial tissue in more severe and prolonged cases. They are such as are met with in all forms of perineuritis—swelling and redness, most distinct opposite the middle of the thigh—thickening of the sheath, invading the substance of the nerve. Microscopical changes have been found, similar to those in acute neuritis elsewhere, in the rare opportunities for observation that have been met with.

The existence of neuritis in all severe cases admits of no doubt; the symptoms—wasting of the muscles and anæsthesia—indicate structural damage to the nerve-fibres, and preclude any other explanation. There is every gradation between these severe forms and those which are slight, and the symptoms in the latter are identical with those of the earlier stage of the severe cases. The early local tenderness proves the initial affection of the sheath and fibrous tissue. All cases of definite persistence and prolonged course are inflammatory. Only transient pains coming and going, associated with neuralgia elsewhere, can be regarded as of neuralgic character. These are often the sequel to a true neuritis, or are, not seldom, the manifestation of a tabetic neuralgia, the pain of which may recur during years in a neuralgic form, and even be influenced in its incidence by change in the weather, so as to receive a rheumatic character.

The symptoms are explained by the morbid process, and the facts of neuritis in general, already described, apply to this disease in every point. It is primarily a perineuritis; the pain in the nerve and its tenderness are due to irritation of the sheath-nerves. The pain referred to the distal portions of the nerve is due to the irritation of its conducting fibres by the interstitial inflammation, while their greater damage explains the sensory impairment and muscular wasting.

The disease, like other forms of irregular neuritis, is not one essentially of the nervous system. It is a local effect of the constitutional malady, gout, or the fibro-rheumatism which results from the inherited diathesis, and each seems to depend on the presence of chemical toxines in the blood, generated in the body by abnormal metabolic processes. Uric acid is probably only one of these, and not the most harmful, or that which has the most special influence. But the nature and origin of these is still one of the most obscure as it is one of the most important problems of pathology, and we must assume that it acts upon tissues disposed to degraded nutrition by the influence of inherited tendency.

DIAGNOSIS.—The distinction between a sciatic neuritis and a sciatic neuralgia is sometimes difficult, although less frequently than might be inferred from current accounts of these diseases, in which the history of the neuralgia has been written from the symptoms of the neuritis. If we recognise that all cases of sciatica with persistent tenderness of the nerve are really neuritic, cases of sciatic neuralgia become extremely rare. The two diseases occur usually under different conditions: the subjects of neuralgia have often suffered from neuralgia elsewhere, and are generally weakly and anæmic. The pain is from the first spontaneous; it occurs as part of more extensive and severe attacks; posture has little influence upon it; movement is not itself painful, although it may excite paroxysms of pain. The pain is referred to the hes and distribution of the nerve rather than to its trunk, or it up or down the trunk; and tenderness of the nerve, if it exists, gether subordinate to the spontaneous pain.

When the nerve is only one of many localities in which the pain is felt, and the patient is an adult, the possibility of its tabetic nature must be remembered. This is practically certain if the knee-jerks are assuredly absent, but of this fact there must be no question. The "lightning" character of the pain is an important diagnostic feature in such cases; although the presence of this character has almost pathognomonic significance, pain of less acuteness and brevity is often significant. It is an instance in which a certain feature has great significance, but its absence is of relatively small value.

Secondary sciatica is usually produced by disease of bone about the hip-joint, or of the joint itself, or by disease in the pelvis. In the former case a careful examination (never to be omitted in any case of sciatica) at once reveals the mischief. When the disease is within the pelvis, and is not primarily connected with the nerves (as the pressure of a growth), the tenderness of the trunk of the nerve is slight in proportion to the pain felt in its distribution; this circumstance should always lead to a careful search for any indication of pelvic mischief. In any case of doubt, or of prolonged course with steady increase in the symptoms, a rectal examination should be made.

Sciatic pain occurs in some diseases of the bones of the spine, in lesions of the cauda equina, and occasionally in disease of the spinal cord itself. In these cases we have little or no tenderness of the nerve; the pain is chiefly peripheral, and very often bilateral. Double true sciatica is so rare that bilateral pain should always suggest disease of the nerve-roots; but the pain in these cases is generally irregular in its distribution, and considerable in other nerve areas in the leg or neighbourhood of the hip, and is often increased much by movement of the spine.

These cases of "secondary sciatica" are of much practical importance, because their nature is usually mistaken; for a long time the patient is thought to be the subject of the ordinary more common form, and is treated accordingly, even when the character of the pain should raise a suspicion of the nature of the case.

PROGNOSIS.—The prognosis of sciatica, not secondary to disease outside the nerve, is good as regards ultimate recovery, but is very uncertain as regards severity and duration. As a general rule, these features are proportioned, but both are influenced by the practicability of adequate rest. Irritating exertion may lengthen the duration of the disease by many months, and indeed relapse may follow each partial recovery for one or two years. Spontaneous pain, and that which is referred to the distribution of the nerves, make the prospect less favourable.

TREATMENT.—The principles of the treatment of sciatica are those of neuritis already described, and only the points of special character need be here repeated. In all cases rest to the limb is important beyond any other measure, and its urgency is proportioned to the acuteness and severity of the symptoms. Many slight cases are converted into severe ones by unwise exertion. Every posture that induces pain, and all movements which increase that which exists, should be avoided. The same is true of pressure on the nerve by a hard seat, and by strong contraction of the flexors cf the knee. Pain may be thus suddenly brought on in severe degree, and may endure.

In more positive treatment the causes of the disease must be remembered, and, among these, the frequency with which it is related to a gouty or rheumatic blood-state. In gouty cases saline purgatives are often of signal service, and are distinctly useful in preventing attacks in those who are liable. In the acute stage of a severe attack, hot linseed-meal poultices should be applied along the course of the nerve. Counter-irritation is of great value, and cannot be employed too early. A commencing attack may often be cut short in a few days by rest, and a series of mustard plasters or small blisters applied over the seats of pain, moved as this changes under their influence, chasing it, as it were, from one spot to another, until it disappears. Internally, whenever there is reason to believe that active inflammation exists, mercury should be given—a grain of blue pill twice daily; nothing else seems distinctly to influence the process. Salicylate of potash or lithia, and nitrous ether, should also be given at the onset—soda salts being avoided. Spontaneous pain can only be relieved by sedatives. Morphia is the surest, but it should only be used for the relief of severe spontaneous pain. Cocaine often proves of great service, if it can be combined with rest, and injected at the actual source of the pain. It should be injected pretty deeply at this place, but not into the nerve. One twelfth of a grain may be first used, increased rapidly to a third or half a grain. It generally relieves, although not in the same degree as morphia, but it seems to have more influence in promoting the subsidence of the inflammation, as described in the account of neuritis, apparently because it has a more distinct influence on the conduction of irritating impulses along the nerves. Morphia, although equally effective elsewhere, may also be injected with advantage over the inflamed part of the nerve, so as to combine the counter-irritation of cutaneous acupuncture with some local as well as general sedative influence. Simple acupuncture along the course of the nerve has been recommended: it gives temporary relief to the sciatic pain, as to any superficial pain, but the cases are very few in which it has a permanent effect. Sedative or counter-irritant liniments and ointments may also be applied along the course of the nerve; the most useful are belladonna liniment mixed with an equal part of chloroform liniment, and aconite ointment, rubbed in until distinct tingling is produced. Electricity is chiefly useful in the later stages; its method of use has been described in the account of the treatment of neuritis. In very obstinate cases, nerve-stretching has done good; sometimes, perhaps, by releasing the nerve from compressing adhesions, but probably more often by effecting an energetic counter-irritation, and enforcing a beneficial rest. It has ceased to be the fashion it was a few years ago, proof of its limited power. Forced movement of the joints, followed by rest, has been sometimes employed. In severe cases absolute rest in bed, and, if necessary, the application of a splint to ensure this, has been recommended. Weir Mitchell* has urged the use of a long splint, reaching from the axilla, jointed, so that the hip and knee-joints may be slightly varied in position, and the combination with this of a soft flannel bandage to the limb. The latter should be reapplied twice daily, from the foot to the groin, to lessen the amount of blood in the limb. If further measures are necessary, the application of the Paquelin cautery to the tender points, or of tin splints filled with ice water, is recommended, and after recovery massage with plentiful feeding.

To prevent recurrence the causes of gout should be carefully avoided, and any signs of a gouty state lessened by an appropriate regimen. Habitual saline aperients are very important; the "flush out" clears away bile that would be reabsorbed, and the result is the same as increased secretion from the liver. Slight pain, threatening a recurrence, should be met by more rest, by counter-irritation with sinapisms, and the careful avoidance of sudden movements, and indeed of all exertion that causes or increases pain. If this is considerable, or the nerve is distinctly tender, complete rest for a day or two may obviate the subsequent need for weeks of rest. If there is the sacro-lumbago that often precedes sciatica, the pain produced by sudden movement may often be lessened by a few moments' rubbing.

General Crural Neuritis.

In rare cases. "rheumatic" inflammation of the fibrous tissue covering the lumbar and sacral plexuses is so extensive that many or most of the nerves to the leg are involved, in consequence of the affection of their coverings at their origin. That of the sciatic may preponderate, as already mentioned in the section on Diagnosis, where the general characters of this form are referred to, and its secondary origin by extension, in some cases of primary sciatic neuritis. The cases in which the affection of the fibrous membranes is extensive give rise to such wide-spread symptoms, pain taking the course of many nerves and accompanied by the tenderness of descending neuritis and impairment of movement, often chiefly from the inhibitory influence of the pain it causes, attended by moderate muscular wasting. There is usually slight increase or decrease in the electric irritability in some group of muscles, with increased knee-jerk, very rarely the reaction of degeneration where the nerves have suffered severely, and then the knee-jerk may be lost if these are the extensors of the knee. In cases of general crural neuritis the extent of the symptoms causes them to simulate such as are caused by disease of the spinal cord,

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* Weir Mitchell, 'International Clinic,' 1891.

[illegible] distinction may be one of real difficulty. But it usually [illegible] aware of its occurrence and general characters, especially [illegible] degree of the objective symptoms, and the existence of [illegible] nerve-trunks near the pelvis. The diagnosis is [illegible] fact that it occurs chiefly in those who present a [illegible] disposition, acquired or inherited. The difficulty [illegible] by the occasional presence of reflected pain on the [illegible] distinguished, however, by its slighter character, and [illegible] to that which is most severe on the side chiefly [illegible] of these cases is often prolonged, and months [illegible] improvement commences, especially when the under- [illegible] state is not recognised, the treatment of which, as [illegible] importance.

[illegible] Neuritis.—Such an affection of the fibrous tissue [illegible] related to a single nerve, or some other branch [illegible] plexus may be the seat of a neuritis like that [illegible] The anterior crural, or a branch, suffers [illegible] resulting symptoms in the front of the thigh, [illegible] These are chiefly sensory; motor symptoms are [illegible] so limited in area.

[illegible] thigh, in the upper two thirds, is a specially [illegible] and occasionally of diminished sensibility. [illegible] sometimes develops in a very chronic manner, [illegible] but curiously enduring symptom, usually [illegible] and apparently the result of a limited [illegible] It seems to have no sinister significance, [illegible] to treatment.

[illegible]MYOSITIS.

[illegible] is perhaps the most convenient by [illegible] usually confined to a single limb, [illegible] symptoms indicating an interstitial [illegible] muscles are the leading feature. They [illegible] character is the pain caused by contrac- [illegible] stimulus which, normally, generates [illegible] fibrous tissue may also suffer, and [illegible] while the condition of the nerve trunks [illegible] symptoms like those of simple brachial [illegible] affection of the nerves is subordinate [illegible] The affection has thus fairly [illegible] to the tissue in which the afferent [illegible] are common to all cases, however [illegible] may spread, and however severe the additional symptoms which partake of the characteristics of other allied diseases. Hence the suitability of the name "neuro-myositis," in spite of some limitation of meaning which this use implies.*

The affection occurs chiefly in patients in the second half of life, and under the same general conditions as brachial neuritis. It is, however, met with more frequently in young persons, sometimes even in children, especially as a troublesome sequel to rheumatic fever, limited to the structures and muscles of the hand. More often it commences in the neighbourhood of the shoulder, in the deltoid and upper arm muscles, and passes down the arm to become more severe in the hand than at the elbow. Slight changes usually occur in the articulation of the shoulder, but more troublesome arthritic adhesions form in the finger-joints and at the wrist, as is common when muscular pain interferes with movements in rheumatic persons. This lasts so long in the intrinsic muscles of the hand, and tissues about the tendons, that the fixation of the joints becomes permanent before there is time for the restoration of the muscles, and some œdema in the acute stage is followed by the characteristic changes in the nutrition of the skin, peeling of the epidermis and wasting of the subcutaneous structures.

But the affection of the muscles is the characteristic feature. The symptoms clearly indicate a rheumatic interstitial inflammation, affecting the tissue in which the afferent nerves begin, and the impulses they convey originate, perhaps, in the mysterious "muscle-spindles." In this tissue the impulses, unfelt in health, are generated by tension and compression, and become painful in some conditions, as after cramp. Then, if the muscle is stretched in extension, or the interstitial structures are compressed by muscular contraction, considerable pain is readily produced. Simple muscular rheumatism, transient, such as the common form of lumbago, seems an affection of this interstitial tissue too slight to persist as permanent inflammation. The chief feature of the form now under consideration is the production of pain by those forms of stimulation to which the nerve-endings normally respond (see p. 58), compression by voluntary contraction, and the passive tension in the antagonist. Pain is caused also in the structures about the joints, especially in the capsules, when the limit of movement is reached which the state of the various structures permits. Even passive movement induces a sudden contraction of the muscles, partly reflex, partly voluntary, which causes pain. This pain spreads widely in the muscles, as those adjacent contract to stop the movement to which the pain seems due, and it is produced with readiness in the morbid state. Hence arises the characteristic feature. Muscular contraction, the great cause of pain, results from any movement, active or passive, and the patient acquires the conviction that no passive movement of the limb is possible without the production of pain in the muscles. Such pain does actually occur whenever an attempt is made to move the joints, as the shoulder and elbow, even in slight degree, or the wrist-joint, if with any suddenness. It is first and most felt not in the neighbourhood of the joints, but in the muscles. Afterwards some may be felt in the structures about the joint. On close and careful observation it will be found that the pain is related much more to the expectation of passive movement than to that which takes place. Before any movement is produced, the muscles contract in order to control and restrict the source of expected suffering. The pain coincides with this contraction, which is unconsciously volitional and reflex, rather than really voluntary. But with the help of some patience and perseverance it is possible to get the limb quite passive, resting without effort in the observer's hands, and to make gentle slow passive movement of the joints without the occurrence of this inhibitory contraction of the muscles. The patient is often astonished at the painless movement of joints when he thought great suffering would result from the least displacement of the parts. But if then a more sudden movement is made, and the alarm and perhaps slight pain cause sudden spasmodic muscular contraction, the pain felt in the muscles is great. There is usually some tenderness on sudden compression of the muscular substance, and this may be considerable and produced with some readiness, but often the sensibility to pressure is inconspicuous. We know, from the state left by common cramp, how easily pain is produced in the muscle by contraction; when this is sudden and considerable the pain may be shared by the nerves; as already stated, those of the brachial plexus above the clavicle and in the axilla, present the same tenderness as is met with in simple brachial neuritis, although in slight and subordinate degree, manifestly from the inflammation of their sheaths. Other structures suffer, as already stated. The result is a considerable amount of fixation of the limb from the pain which is produced by movement; this has more than one source, and it may be difficult at first to discern that from which the chief suffering proceeds. The arthritic changes within the joints produce adhesions which, with the alteration in the capsule, fix the joint in an abnormal posture, generally that which falls short of complete extension. The contraction of the muscles to inhibit movement tends to be more and more persistent and continuous, and this state of rigidity is prone to cause some shortening of their structures; nutritional change fixing the functional states assists in producing the slight deformity and fixed posture of the joints that are frequently observed.

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* It has indeed been proposed by Senator to use it to designate another disease, the rare acute symmetrical myositis (described further on), which is like multiple neuritis in toxic causation, and distal bilateral distribution ('Deut. med. Wochenschr.,' 1893). The term has not, however, come into use for this affection, and seems not needed for it. "Acute myositis" or "polymyositis," the names employed in Germany, are sufficiently adequate to leave to its present application the term "neuro-myositis."

The malady is commonly of very prolonged duration, developing insidiously even after recovery from an initial, more acute attack of local rheumatism, especially when slight persistent pain on movement is maintained by the neglect of needed rest. The affection is usually unsymmetrical, affecting one side only, after the fashion of these rheumatic affections of the fibrous tissues, of sciatica, or of brachial neuritis. Nevertheless, in moderate typical form, the manner in which it presents itself as an affection of the interstitial tissue of the muscles and their nerves justifies the view which is expressed in the designation given to it; and the peculiar implication of the afferent muscle-nerves, in consequence of its interstitial localisation in the muscles, justifies its inclusion among the maladies here described.

This is further warranted by the condition which commonly results. The duration of the affection and the changes which ensue in the substance of the muscles and the tissue about the joints, and the fixation of these during the time that elapses before the cessation of pain permits freedom of movement, by that time mechanically hindered, bring the cases into the class of residual consequences of nerve lesions.

The resulting interference with use of the part is greater than is proportionate to the amount of rheumatic inflammation, being especially great when much sensitiveness has proceeded from the long-continued pain, and fear of its induction outlasts the danger and exceeds it in degree. Some chronic neuralgic pain is apt to remain, due in part to the predisposition to neuralgia which most patients of this class possess.

Another curious sequel hinders the return of mobility in these cases. Passive movement is difficult out of proportion to any pain produced, although this results if the attempt is persevered in and its strength increased. The common effect is a peculiar rigidity of the limb, such as at first suggests firm adhesions at the joints—an impression that may be easily retained if the examination is hasty. Its nature is rendered clear by sufficient patience and close observation; passive movements must be attempted with the utmost gentleness and slowness, and the attention of the patient turned to some other part during their course, while the observation of the examiner remains fixed.

The same care is necessary in local therapeutic measures if these are to be successful. Gentle upward rubbing to the muscles should be afterwards combined with slow passive movement, at first scarcely appreciable in degree, and continued with the endeavour to keep behind the production of the muscular spasm. This will often permit good to be effected by local applications of heat, dry or moist, liniments, &c. The internal treatment needed is such as is suitable for the gouty and rheumatic constitutional state, with tonics where these are indicated, and is of the same general character as that recommended for brachial neuritis.

Gout and Rheumatism in the Causation of Neuritis.—It may seem to many readers that the range of influence of these constitutional states has been pushed too far in the preceding account of the affections, which are primarily of fibrous tissue, and only of the nervous system by reason of proximity. The difficulty may be increased by the number of patients who present no adequate personal or hereditary evidence of such causal disease or tendency. But it should be noted that the influence of gout, including ancestral gout, is a subject on which the young practitioner starts with a high degree of scepticism regarding the teaching of his seniors. But year by year his doubts become fewer, as they are rubbed away, or removed more sharply, by contact with fact.

The negative influence of the cases in which the cause cannot be traced, or only in slight degree, should be carefully weighed before the result is placed in the scale.

MULTIPLE NEURITIS.

The term "multiple neuritis," or "polyneuritis," is applied to the condition in which many nerves are inflamed simultaneously or in rapid succession. This multiplicity is combined with bilateral symmetry, alike in its seat and in the special character of the symptoms. These constitute its most obtrusive features. If only a few nerves are involved, the symmetry becomes a more salient feature than the multiplicity. The symmetry is not only characteristic, it is of profound significance, because commonly combined with a third feature, the fact that the affection is most intense at the extremities of the nerves, and lessens progressively towards the centre. This "peripheral" symmetry can only be due to a cause circulating in the blood or inherent in the vitality of the nerves. The former is the common mechanism, but some influence of the latter is indicated by this seat,—farthest from the central source of nutritional energy, where the resistance to morbid influences is least.

On account of the seat the disease has also been termed "peripheral neuritis," a name that is accurate but somewhat confusing, because it has been used to distinguish all affections of nerve-trunks from central diseases causing similar symptoms, especially in the case of the cranial nerves. It should not be employed in this sense.

The name "polyneuritis" has also come into extensive use, and perhaps will more readily secure association with another important characteristic, though practically identical with "multiple neuritis." The microscope shows that the changes are almost confined to the proper tissue of the nerve-fibre, and do not implicate the sheath or interstitial tissue, as a rule, even when acute. This, indeed, we might expect from the special limitation in the function deranged. But it affords a contrast to the forms of neuritis previously described. These may be multiple, and even, very rarely, symmetrical, as when both sciatic nerves are inflamed. But the symmetry is rough, and it does not extend to precision in the function disturbed, and soon ceases to be distinct. It is this affection of the true nerve tissue, and its results, which marks the symmetry of the form under consideration, and is absent in the occasional multiplicity of the inflammation met with usually in single nerves. This character, of absolute ultimate importance, is more likely to become attached to "polyneuritis." A name is needed which embodies also the idea of symmetry.

The discovery that certain combinations of symptoms, formerly thought to depend on disease of the spinal cord, are really due to disease of the peripheral nerves, is one of the most important steps in the recent advance of pathology. It has profoundly modified many of our conceptions, and has opened a new field in pathology, the cultivation of which has but begun.

The occurrence of multiple neuritis was first demonstrated by Duménil, of Rouen (1864), although the leprous form (which differs in important points) had been previously described by Virchow. Todd also deserves credit for a correct and clear perception of an affection of the nerves as the cause of lead palsy in 1854.[*] Graves, indeed, long ago suspected that many cases of paralysis were due to disease of the nerves, but he based his opinion on the normal aspect of the spinal cord; and in most of the cases he describes it is probable that modern methods of examination would have revealed disease. Symptoms so peculiar had been noted from time to time; first, perhaps (1822), by Dr J. Jackson, of Boston, U.S.A. They were fully described by Duchenne in 1858. Even Duménil's observations attracted little notice, and it was not until fresh facts were brought forward by Joffroy (1879), Leyden (1880), and Grainger Stewart (1881), that attention was generally directed to the subject. Since that time a very large number of observations have been published, and the wide relations of the morbid process are now perceived.

Among these one stands out conspicuously. The forms of multiple neuritis which present local and functional symmetry and are of parenchymatous nature must be ascribed to a blood-state having equal access to all parts, but a special action on those that suffer, or do so most readily, owing to a peculiar and constant disposition inherent in those structures thus to be affected. The cause of this affection is the presence in the blood of some virus, and the salient fact is that the toxic agent is usually a poison of chemical nature, a metal, or an organic chemical material often resulting from the growth of organisms in or out of the body, sometimes apparently from disordered processes of digestion or excretion, or deranged metabolism within the body, varied in seat and nature. To such a poison some nerve-fibres are specially susceptible, just as they are to curara, or to and the distinction may be one of real difficulty. But it usually suffices to be aware of its occurrence and general characters, especially the moderate degree of the objective symptoms, and the existence of tenderness of some nerve-trunks near the pelvis. The diagnosis is also aided by the fact that it occurs chiefly in those who present a gouty or rheumatic disposition, acquired or inherited. The difficulty may be increased by the occasional presence of reflected pain on the opposite side, distinguished, however, by its slighter character, and correspondence in seat to that which is most severe on the side chiefly affected. The course of these cases is often prolonged, and months may pass before improvement commences, especially when the underlying constitutional state is not recognised, the treatment of which, as in sciatica, is of primary importance.

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[*] The first ascription of a form of paralysis to this lesion was by Dr. Todd, in the case of lead palsy. "I believe that the muscles and nerves are early affected, and that at a later period the nerve-centres become implicated. The nervous system is thus first affected at its periphery, in the nerves, and, the poisoning influence continuing, the contamination gradually advances towards the centre" ('Clin. Lect.,' 1854, p. 9).

Anterior Crural Neuritis.—Such an affection of the fibrous tissue may be limited to that related to a single nerve, or some other branch of the sacral or lumbar plexus may be the seat of a neuritis like that which underlies sciatica. The anterior crural, or a branch, suffers most frequently, with resulting symptoms in the front of the thigh, or in the outer part. These are chiefly sensory; motor symptoms are seldom conspicuous in cases so limited in area.

The outer part of the thigh, in the upper two thirds, is a specially frequent seat of pain and occasionally of diminished sensibility. Anæsthesia in this region sometimes develops in a very chronic manner, without pain, as an isolated but curiously enduring symptom, usually stationary when discovered, and apparently the result of a limited neuritis of chronic form. It seems to have no sinister significance, but has little tendency to yield to treatment.

NEURO-MYOSITIS.

The term "neuro-myositis" is perhaps the most convenient by which to designate an affection, usually confined to a single limb, generally the arm, in which symptoms indicating an interstitial rheumatic inflammation of the muscles are the leading feature. They become tender, but the especial character is the pain caused by contraction of the fibres, *i. e.* by the stimulus which, normally, generates afferent impulses. The adjacent fibrous tissue may also suffer, and especially that about the joints, while the condition of the nerve trunks may be such as to give rise to symptoms like those of simple brachial neuritis; but in such cases the affection of the nerves is subordinate to the peculiar state of the muscles. The affection has thus fairly well-defined features related to the tissue in which the afferent impulses originate, and these are common to all cases, however widely the rheumatic affection may spread, and however severe the besides giving rise to the product, perhaps a chemical substance, comparable to alcohol, capable of causing a parenchymatous neuritis. The two forms may then be conjoined in a puzzling manner, varying in relative amount in different cases. It is probably thus that the forms of diffuse or total neuritis are produced, occasionally met with in diphtheritic paralysis. We have here a field still to be investigated.

Not less important and mysterious are the distinct indications that symmetrical neuritis results from toxines developed within the body from the effects of other chronic diseases. Diabetes is one of these, but the poisonous agent is not the sugar in the blood, for the palsy occurs when there is but a very small amount in the urine. One deranged chemical process involves others, which vary in different persons, and minute chemical variations may determine profound differences in effect, ranging from the innocuous to the toxic. In advanced organic kidney disease also signs of polyneuritis may appear—pains in the legs, loss of the knee-jerk, paralysis of the muscles, tingling of the skin, anæsthesia. Poisons capable of producing the condition, but of unknown nature, seem sometimes to be taken in from the outside, although we know not how, for cases of characteristic features occur when the most exhaustive search fails to discover a known cause.

Lastly, symptoms of this state, moderate in degree—weakness at the ankle, loss of the knee-jerk, subjective sensations, or definitely defective sensibility below the knees,—sometimes come on gradually in the old, without other cause than the influence of advancing years. The symptoms seem not to be progressive, and become less noticed when they are slight, although they are sometimes maintained by attention in a troublesome degree. They are apparently due to a degenerative tendency due to age, and are signs of a senile form. We must assume that the nerves which suffer most in this disease possess less power than others of resisting morbid agencies, and that their capacity for vital endurance varies in different individuals, to understand the occurrence of the malady in those who suffer, and the greater readiness with which it is caused in some by a small quantity of the poison. Indeed, we are confronted at the outset by a fact which involves such a difference inherent in the two sexes. The most common cause, alcohol, gives rise to polyneuritis far more frequently in women than in men. Half a dozen cases will be met with in females before one is seen in a man. An excessive degree of such a disposition to degenerate would necessarily involve an earlier manifestation of the imperfect nutrition which is the normal termination of life.

The discernment of this symmetrical derangement of function, of its dependence on the nerves, and the action on them of morbid agents conveyed by the blood, has enlarged our pathological conception by revealing the nature of cases previously misconceived, because absolutely not common, and differing too widely in those features that would be put together for their nature to be readily recognised. Indeed, their recognition is probably one of the results of the perception of how much is meant by the disappearance of the knee-jerk, and the need to observe minutely and compare carefully all cases in which it disappears. This was at least one cause for the appreciation of the significance of the isolated palsy of the flexors of the ankle coinciding with such loss as it was found to do, and the more careful study of such cases to which the observation led.

But the discovery of the relation of the affection of the nerves to alcohol, which swiftly followed the perception of its peculiar features and the character of their cause, has had effects of even greater importance, and an influence on our discernment still far from exhausted. Alcohol is a purely chemical agent. Various metallic poisons, acting in minute amount and slowly, were also found to have similar effects, while in other cases the toxines seemed to be also organic chemical substances. This was also supplemented by the discovery that derangement of the nervous system such as occurs in tetanus was due to substances of similar nature, resembling strychnia in effect (a vegetable alkaloid), and yet generated by organisms in the system; that the cause of diphtheritic palsy was also a chemical substance, a modified albuminose; and that a similar agency is the cause of the polyneuritis which may follow other acute specific diseases. A wide, unstudied field of chemical pathology has thus been opened, on which multiple neuritis will find a place, and clearly included therein are the forms which result from disordered processes within the body in the subjects of gout, exposure to cold, and the like. Chemical change in the nerve-tissue attends all its functions, and by their activity the renewal of elements is increased. Its character depends on the composition of the plasma bathing them, and containing the molecules to replace those broken up in function. A slight difference in the constitution of the replacing molecules seems merely to derange the process at first, and a trifling alteration in function is the only result, such as subjective tingling when the sensory nerves suffer chiefly, or lessened perception of the most gentle stimuli. The persistence of the process leads to graver change in molecular composition and more serious effects, and at last the change amounts to an alteration which may abolish function and be visible under the microscope. Such, for example, is the effect of arsenic; typical polyneuritis results from the constant presence of a definite quantity in the blood. Arsenic has chemical correspondences with phosphorus, as well as differences, which enable us to understand its assimilation by the nerves, and also the gradual changes that result, molecular at first, but at last structural. The affinity for certain parts of the nervous system presented by different substances enable us also to understand that acute arsenical poisoning may be followed by a similar chronic neuritis of bilateral and peripheral distribution, and both motor and sensory symptoms, while very chronic poisoning uses more various effects, apparently determined by dose and the mbinations which the state of the system causes it to form, and in which it is presented to the nerves. The specialisation of the sensory symptoms is remarkable in its variations from all causes; one form of sensation may alone be disturbed. Always, however, the form is the same, and the seat is the same on each side; and this feature, whether the disturbance is trifling or grave, should arouse a suspicion that we have to do with the action on the nerves of a toxic agent in the blood. The importance of this indication cannot be insisted on too often or too strongly. It will often save from serious error, from loss of precious time, and the patient from much suffering and prolonged disability.

In both acute and chronic forms the influence of the toxic agent may be exerted on the central organs as well as on the peripheral nerves, not only so as to give rise to more or less diffuse inflammation, but limited degeneration of tracts of fibres, as of the posterior columns or the pyramidal tracts. The frequent relation of the former to a toxine is well known, and degeneration of the lateral tracts is known sometimes to begin at the extremities of the fibres in a way analogous to that of the peripheral nerves. It is not, therefore, surprising to find central symptoms combined with those of neuritis in various forms, and the resulting effects may present characters of a puzzling aspect. Such wider distribution must be ascribed to the predisposition of the subject and the character of the active agent. It is indeed surprising that the effects of this one, so frequently limited as they seem to be, and the common restriction of their incidence, are significant of a definite uniform difference in the chemical relations of the nerve-structures, no doubt with an absolute relation to function under normal conditions as well as to disease. Indeed, the physiological importance of the facts of toxic neuritis are most distinct. They suggest that chemical processes have a larger share in determining those of vital function than has been thought, and that their range may be found more extensive and important than was previously suspected.

An important question has been raised by the discovery that the central structures often suffer in cases of peripheral polyneuritis, and that some toxic agents seem to act also on the nerve-cell, the central element of the neuron, as well as on the peripheral structures. But the facts are still too scanty to be of use, and will be referred to again in the section on pathology.

It is to the pathology of the disease that we must look for its effective subdivision, but our knowledge of the subject is still imperfect. The causes of polyneuritis, numerous and various as they are, still furnish the best ground for a practical classification of the forms of the disease such as will assist us in recognising them and in treating them.

Symmetrical regularity means a blood-state acting alone; irregular multiplicity generally means a constitutional state combined with local determining causes. When very few nerves are affected irregularly, the general influence sinks to the level of a predisposition, and this is not always to be recognised when a single nerve is affected.

ception of how much is [illegible]
and the need to obser[illegible] has also been made a ground
which it disappears. Th[illegible] much in aspect and grouping
of the significance of [illegible] chronic the process the more
coinciding with such los[illegible] and of its effects on function.
study of such cases to [illegible] in the nerve-elements resembles

But the discovery of [illegible] been termed "degenerative
alcohol, which swiftly foll[illegible] with cases both acute and
and the character of their [illegible] the toxic agent.
ance, and an influence [illegible] anomalies until we know more of
Alcohol is a purely chem[illegible] which the varieties are produced;
in minute amount and [illegible] may survey their relationship, the
while in other cases the [illegible] groups.
substances. This was [illegible] them first generally; some will
rangement of the nervo[illegible]
due to substances of [illegible] of a known poison, a substance
vegetable alkaloid), and [illegible] simplest is—
that the cause of diphth[illegible]
a modified albuminous [illegible] most frequent cause. Other or-
the polyneuritis which [illegible] outside the body, comparatively
wide, unstudied field of [illegible] in a similar manner.
which multiple neuritis [illegible] blood, the precise nature of which is
are the forms which res[illegible] a chemical substance formed within
in the subjects of gout [illegible]
change in the nerve-t[illegible] of a specific disease, probably due
activity the renewal of [illegible] body of the organisms of the
on the composition of [illegible] symptoms of another class are due
molecules to replace tho[illegible] Hence the polyneuritis which
in the constitution of the [illegible] fevers. A poison having similar
the process at first, and [illegible] organisms of septicæmia in its varied
result, such as subjecti[illegible] causing most of the cases of puer-

chiefly, or lessened perc[illegible] of the chemical processes of the body
sistence of the process [illegible] capable of acting on the nerves. I[illegible]
tion and more serious [illegible] of the derangement that manifests it
alteration which may [illegible] but other abnormal substances [illegible]
microscope. Such, for [illegible] readily detected, or give rise to other
polyneuritis results from [illegible] Thus peripheral neuritis occurs i[illegible]
in the blood. Arsenic ha[illegible] excess of sugar in the blood, bu[illegible]
as well as differences, wh[illegible] substance the nature of which is n[illegible]
by the nerves, and also th[illegible] have a disorder of the chemical p[illegible]
first, but at last structur[illegible] an excess of one substance without oth[illegible]
nervous system presented [illegible] abnormal in their nature or in the
understand that acute [illegible] may be known the characteristics of so[illegible]
similar chronic neuritis of [illegible] the neuritis is an apparent consequence;
both motor and sensory [illegible] causing no distinct effects, and t[illegible]
causes more various effect[illegible] the only consequence of the causal derang[illegible]
combinations which the sto[illegible] may be termed secondary and prim[illegible]
[illegible] according as other abnormal products ma[illegible]
[illegible] or are latent, but there is evidently
[illegible] between the two classes.

(c) *Rheumatic.*—An important variety of this class is that in which a toxine, seemingly of corresponding nature, results from an external influence, such as exposure to cold.* Rheumatic polyneuritis is a special form, and yet seems to be a metabolic form acutely excited by a process the occurrence of which we can see, but cannot understand. It varies much in its intensity, and in the severity and character of the attendant catarrhal or rheumatic symptoms. They may amount to those of actual accompanying disease, which may assume predominant intensity and bear the aspect of a primary affection to which the neuritis seems to be secondary, and a consequence rather than a mere concomitant. But its cause is evidently the presence of an agent in the blood capable of deranging acutely the nutrition of the nerves in a manner more or less grave and enduring. The morbid agent thus generated must be produced by a disordered action occurring under the influence of the exposure, but in consequence of a pre-existing state of the system, which we can best conceive as the presence in it of some morbid state, most probably of the nature of a chemical material, abnormal in character, although perhaps differing but little from those that are normally present. It may be more closely allied to such products, although we conceive it most readily as analogous to a simple poison.

(d) *Septicæmic.*—From such cases we pass to others in which the cause seems to be more special in nature, and in which the resulting disorder of the peripheral nerves is part of the general disturbance of septicæmia. Septicæmic polyneuritis constitutes an important variety, frequent and often most grave. Its causes are numerous, but present two important variations in nature; the cause may be an injury or operation, or it may be of entirely internal nature. An infective process is the source of an organismal mechanism in many cases of this class. To it belong most, perhaps all instances of puerperal polyneuritis, an important group, which vary in nature, however, so as to make their precise classification very difficult. They present the common symptoms in characteristic and usually in acute form, but these may begin before or after delivery in association with other symptoms of blood-poisoning although they predominate in the cases that can properly be placed in this category. But variations in course are sometimes conspicuous, and cases are seen which present no evidence of distinct causation. Without doubt, however, the majority are due to some form of septicæmic process, and this is true when other signs of it are inconspicuous. Puerperal polyneuritis is septicæmic as an almost invariable rule. Rare cases are met with, however, in which the symptoms of neuritis develop insidiously, and it may be some time before delivery, in a manner that increases their mystery, while this is further augmented by the termination of these cases in recovery.

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* See Ross and Judson Bury's 'Treatise on Peripheral Neuritis.'

The acuteness or slowness of the [illegible] disorders
of distinction, but the symptoms [illegible] although its
according to their intensity. The [illegible] outside or
perfect is the limitation of the [illegible] the pathology
In the most chronic form the [illegible], in which
a pure degeneration, and hence [illegible] agents of the
neuritis." From most causes we [illegible] as largely
chronic, according to the intensity [illegible] a chemical agent

No arrangement can be free from [illegible]. The indirect
the causes and of the mechanism [illegible] acts on the nerve-
but provisionally, therefore, that [illegible] strong when the
cases may be placed in the following [illegible] that such an agent.

It will be instructive to survey [illegible] of tetanus by
need afterwards more special [illegible] that in each instance

I. Toxic: due to the presence in [illegible] in which no ex-
commonly so described; of [illegible] nutrition fails spon-
(*a*) ***Metallic:*** lead, silver, [illegible] in the important
(*b*) ***Non-metallic,*** as [illegible] come on in late life,
ganic substances, [illegible] of the nutrition of
simple and well [illegible] a general constitution a[illegible]

II Toxæmic: due to some virus [illegible] direction for som[illegible]
unknown, but which is [illegible] we cannot discern. I[illegible]
the body. It may be— [illegible] effect. It is manifest[illegible]
(*a*) *Pathogenetic,* the [illegible] and chiefly as irritat[illegible]
to the growth [illegible]
disease, as poisons [illegible] degeneration except as
to the organisms [illegible] as a condition that is [illegible]
may succeed the [illegible], and we must regard so[illegible]
effects results from [illegible] of the morbid state
forms, traumatic [illegible]. Yet the fact that s[illegible]
peral neuritis. [illegible] frequently as facts show i[illegible]
(*b*) ***Metabolic.***—A [illegible] to an idiopathic failure
may give rise to [illegible] constitute the morbid sta[illegible]
may be the only [illegible] it plays any considerable
presence by [illegible]
formed which [illegible]
obtrusive [illegible] more detailed consider[illegible]
diabetes, not [illegible] due to alcohol is the
from some other [illegible] manifestation of the d[illegible]
yet known. [illegible]
cesses that [illegible] symmetrical bilatera[illegible]
materials being [illegible] of many nerves,
amount. There [illegible] single nerves, but s[illegible]
malady, of [illegible] a result of the dire[illegible]
they may be [illegible]. True acute [illegible]
neuritis develop[illegible] in some cases.
ment. These [illegible] produced by the or[illegible]
metabolic [illegible] polyneuritis must [illegible]
fest their [illegible] met with in [illegible]
essential [illegible]

suggesting that a toxine may be produced at an early stage of the disease, capable of acting on the nerves and causing polyneuritis. The subject is considered in the section on Locomotor Ataxy, but it is important to mention it here, because this disease presents an example of central and peripheral lesions due to the same cause, and because an affection of the sensory nerves almost identical in distribution and nature, and giving rise to similar effects, is sometimes the consequence of alcohol, and occasionally also of other agents, as arsenic.[*]

Tubercular polyneuritis is a form regarding which we have still much to learn. It has occasionally developed in the course of phthisis, and has been found to be of the purely parenchymatous or degenerative variety. Severe cases, with characteristic motor and sensory symptoms, in which other causes, such as alcohol, can be excluded, are rare, but their occurrence is well established,[†] and they seem to justify the conclusion that the tubercle bacillus is capable of producing a poison having a specific action on the nerves—a mechanism also indicated by the fact that the bacilli themselves cannot be found in the affected structures. It is said that similar slighter neuritis is frequently to be found after death in cases of phthisis, and probably produces symptoms that are overlooked in the general prostration of the later stages of the disease. Some severe cases recorded as tubercular are, however, not free from the suspicion that alcohol was in part or wholly the cause of the neuritis. There is, moreover, another relation between multiple neuritis and phthisis; the latter often develops in the course of alcoholic neuritis, and is a frequent cause of death.

Gout gives rise to interstitial neuritis and perineuritis, usually isolated, but sometimes multiple and then irregular in distribution. Yet chronic alcoholism occasionally coincides with the gouty state, and may cause a combination of the symmetrical toxic form of typical character, and an isolated neuritis, such as inflammation of the sciatic nerve—a combination which may puzzle unless this origin of it is recognised.

Most *Acute Specific Diseases* have been followed by neuritis, some more frequently than others, especially diphtheria, variola, measles, typhus and typhoid fevers; and influenza has had this sequel with notable frequency during the epidemic of the last five years. The interval that usually elapses between the acute disease and the neuritis gives strong support to the pathology of the disease above described. The occurrence of the neuritis does not seem to be related to the severity of the primary disease, but rather to some peculiarity in the special organisms, such as so often determines peculiar features in an epidemic. This feature is conspicuous in all the acute specific diseases. In one case, which developed six weeks after varioloid, with pains in the limbs, atrophy of the arms and legs, &c., the purely neuritic nature of the morbid process was ascertained six months later, when the patient died from pneumonia. But the mechanism is apparently more complete in some maladies. In diphtheria a poison is produced which has a more extensive action on the nerve-structures. This is described in more detail in the second volume. In this disease, moreover, the acute stage is attended by an acute inflammation of all the elements of the nerve-structures, and not merely of the functional parts—a more intense morbid agent being apparently active. Malarial neuritis seems to occupy a position intermediate between the secondary and primary forms, since it sometimes follows malarial fever, and sometimes develops without previous fever in those who have been exposed to the influence of malaria. We have much still to learn regarding the pathology of these forms.

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[*] Durdufi ('Neur. Cent.,' 1893, p. 632) directs attention to a fact, especially insisted upon by Leyden, Gowers, and others, that gonorrhœa may give rise to morbid conditions of the nervous system, and mentions especially neuritis and myelitis.

[†] See Oppenheim, 'Zeitschr. f. klin. Med.,' 1886, p. 230.

Septicæmic polyneuritis is a rare variety, but one of the most formidable in many cases, the nerves to vital organs suffering with relative frequency and severity. The most severe cases have been traumatic, the result of organisms introduced from without into some wound, or of some injury or local inflammation capable of giving rise to blood-poisoning. In some such cases the disease has been unaccompanied by the usual symptoms of septic blood-states, but has followed its cause at an interval that suggests, taken with other analogous facts, that it is due to some virus produced probably by septic organisms of a special character. For instance, a man, thirteen days after a stab-wound beneath the clavicle, which healed well, had an attack of parotitis with facial palsy. On the fortieth day there developed paralysis of tongue, vocal cords, and limbs, and on the sixth day after the onset of these symptoms he died from respiratory palsy. Extensive peripheral neuritis was the only nerve lesion.* In a less severe case recorded by Barrs† the symptoms commenced three weeks after a neglected wound of the hand, which produced inflammation of the lymphatics, extending up the arm to the axilla. The symptoms of neuritis began in the injured limb, and invaded successively the other extremities, causing characteristic weakness in the lower limbs, with loss of the knee-jerk. He slowly recovered. It is possible that some cases of this class result from unsuspected foci of inflammation within the body, in which pus becomes pent up and causes the blood-state.

In cases with a possible internal source of blood-poisoning, polyneuritis may develop as an apparently primary disease if the source of the septicæmia has been undiscovered. The nature of such cases is often mysterious. They are not separable from those due to deranged metabolism, or to some toxine arising by a different mechanism.

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* Roth, 'Corr.-Bl. f. Schw. Aerzte,' 1883, No. 13.

† 'Amer. Journ. Med. Science,' Feb., 1889.

Other primary cases occur, moreover, in which there is no cause for septicæmia, and yet of such severity as to suggest a poison coming from a powerful external source. The only explanation we can give of these is that they are probably due to a virus received from without, the precise nature of which is unknown, and that there are sources of such toxines of which we are still ignorant. Some cases have been met with in which the symptoms of multiple neuritis, perfectly characteristic, came on without any discoverable cause, and ran a course in most cases mild, and ending in recovery; in one or two others severe, and ending in death. Some cases of what is popularly termed "blood-poisoning" are probably of this nature. A condition also occurs during the puerperium, and is probably the result of some accidental septicæmic process. The type is one of severe polyneuritis.[*] A similar condition has also been described[†] as occurring during pregnancy, the symptoms all disappearing after delivery. If certain drain poisons, probably organismal, are capable of giving rise to a disease, diphtheria, of which multiple neuritis is often part, there is no improbability in some forms of virus, of similar character and analogous source, having such neuritis for their chief effect.

In this connection it is instructive to note that an epidemic is on record in which paralysis preceded the throat affection (see Diphtheritic Paralysis, Vol. II). As an instance of this primary form may be mentioned the case of a medical practitioner who was attacked with obstinate vomiting and diarrhœa succeeded by constipation, for which no cause could be discovered, quickly followed by "numbness" in the hands and feet, and a difficulty in using them. When I saw him, a fortnight later, there was loss of tactile sensibility, limited to the palms and soles, weakness of the legs, and no knee-jerk. The weakness rapidly increased and extended, and at the end of five weeks from the onset he died. Such a case presents all the features of the toxic form.[‡]

The *Rheumatic* form embraces all the cases of polyneuritis that are due to exposure to cold. Rheumatic fever, due to a similar exposure to cold, must depend on a morbid blood-state, produced in some way by the disturbing influence of the chill on the metabolic processes within the body. The poison thus generated must vary much in its

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[*] Möbius, 'Munch. med. Wochenschr.,' 1892, Bd. xlv; Bernhardt, 'Deutsch. med Wochenschr.,' 1894, Bd. 1; Eulenburg, 'Deutsch. med. Wochenschr.,' 1895, Bd. viii, ix; Sottas and Sottas, 'Gaz. des Hôp.,' 1892; Turney, 'St. Thomas's Hosp. Reports,' vol. xxv, 1898, who describes three varieties: (1) generalised neuritis, (2) localised neuritis affecting (*a*) upper, (*b*) lower limbs, and (3) neuritis affecting a single nerve without any ascertainable local cause.

[†] Stiefel, 'Neur. Cent.,' 1893, 352.

[‡] It should be mentioned that other circumstances excited a suspicion of intentional chronic poisoning. This fact does not lessen the suggestiveness of the case. Multiple neuritis must henceforth constitute an element in many questions of medical jurisprudence. Its discovery might give greater weight to indications of some forms of poisoning.

[illegible] the acute lesions which [illegible] Vol. II. Hence it is not surprising that the [illegible] in some persons, be such as to cause a [illegible] multiple neuritis, symmetrical and [illegible]. In a case recorded by Putnam, fatal in seven days [illegible] muscles, following directly exposure [illegible] swelling of the spleen such as is met with in typhoid [illegible] states. Two cases of fatal septic polyneuritis [illegible] by endocarditis are recorded.* We do not know to what these variations are due. In some there has been a previous [illegible] to chronic alcoholism sufficient to explain the extreme degree of the resulting process. The causes of the differences in the results are, however, still involved in mystery. From this, as from other causes, but perhaps with greater frequency, the blood-state is apt to give rise to more irregular central inflammation, and the two may coincide. The connective tissue of nerves is, moreover, of the same nature as that on which the rheumatic poison exerts its special influence, and the modes of onset of sciatica have shown us that the rheumatic process may pass from the fasciæ to the nerve-sheaths. A consideration of the facts makes it probable that cold may cause (1) an isolated adventitial neuritis; (2) a multiple neuritis of the same character, irregular in distribution, probably by the agency of a blood-poison related to that which causes muscular rheumatism and the affection of the fibrous tissues of the joints in rheumatic fever; (3) a symmetrical parenchymatous polyneuritis due probably to a special blood-state of greater specificity than that which produces the adventitial form, and perhaps allied to that which gives rise to pneumonia and to cerebro-spinal meningitis. Pneumonia, at least, may be combined with multiple neuritis as a result of the same exposure.

The diabetic form presents many varieties, and our knowledge of it has been already referred to so far as its imperfect character [illegible]. It is not related to the amount of sugar in the urine—in many of the sufferers this has been small (in one, indeed, as low as one half per cent.); nor is it readily influenced by a dietetic reduction of the amount of sugar in the blood. Hence, as we have seen, it is supposed to be due to some toxic product that is formed in the perverted metabolism of the diabetic subject, comparable to acetone, although certainly not acetone itself.†

Diabetic polyneuritis is the form most frequently met with, at any rate so far as well-marked cases are concerned; this form preponderates in most countries over all others put together, but at present many of the slighter sensory forms escape recognition, and the preponderance will probably be much less when all cases are recognised. It results chiefly from the stronger forms of alcohol, especially from spirits. It is more common among those who take small quantities frequently than among those who indulge in an occasional "bout" of intemperance, probably because the total quantity consumed is greater. Strange to say, it is far more frequent among women than among men, probably at least three times as frequent. The cause for this is difficult to discern. On the one hand continuous drinking is more common among women than is occasional intoxication; and women who take alcohol to excess generally do so in the form of spirits, while men often take it in the form of beer. At the same time the opposite proclivity is seen in the case of delirium tremens, and it would seem as if there were a difference in the nervous system of the two sexes, whereby a susceptibility of the nerves in women replaces that of the brain in men.

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* [illegible] 'Trans. Am. Neur. Assoc.,' 1892.

† Williamson ('Med. Chron.,' Nov., 1892) found in fifty cases of diabetes that the knee-jerk was absent in 38 per cent., and obtained only with reinforcements in 12 per cent.

The amount of alcohol habitually taken, and the duration of the habit before the onset of the affection, vary much. It has been thought that a neuropathic disposition renders a less quantity and briefer period adequate. Other causes often co-operate with alcohol in exciting the disease, especially exposure to cold, and, in the lower classes, insufficient nourishment. Exposure is especially effective in those in whom slight symptoms exist but attract little attention, and the condition of the nervous system is, as it were, prepared for a severe outbreak.

Idiopathic Forms.—Regarding these varieties of idiopathic polyneuritis, little remains to be added to what has been stated at p. 146. The evidence rests chiefly on the post-mortem discovery of the changes in the nerves, but it is probable that more careful attention to the point will show that symptoms are not unfrequent which now receive little attention, or are regarded as functional only. More attention is also needed by the cases in which a neuropathic tendency or some depressing emotion seems to be the cause of this disease, as they may be of the corresponding degeneration of the pyramidal fibres within the cord in "lateral sclerosis," a degeneration which also begins at the extremity of the fibres and extends upwards.

Atheromatous Neuritis merits separate recognition on account of its peculiar mechanism. In these cases the nerves of the limbs have been extensively damaged through atheroma (or "arteritis obliterans") of their arteries. Necrotic inflammatory processes take place in the parts of the nerves supplied by the affected vessels, and there result symptoms that resemble those of the toxic form in character, although differing in the greater irregularity of distribution. The symmetry that constitutes so striking a feature of the common cases is absent, but arterial degeneration may be symmetrical, and an incomplete rough symmetry may thus result. It is conjectured that such disease of the nerves will be found to be frequent in the cases in which senile disease of the arteries gives rise to gangrene, and that it is partly through the agency of the neuritis that the gangrene is produced; but this view overlooks the potent influence of the arterial obstruction itself.*

General Etiology.—Multiple neuritis is a disease of adult life. The only form met with in children is that which sometimes accompanies polio-myelitis, and may perhaps now and then be met with apart from the spinal malady, as an infantile variety of multiple adventitial neuritis, irregular in distribution. The other common forms occur chiefly between twenty and fifty; the alcoholic between thirty and forty twice as frequently as in either the previous or later decade, but it may be met with up to sixty years of age; while the rare senile (purely degenerative) variety, and the still more rare arteritic form, are met with at still later ages. Females constitute a majority of the cases, the degree of which cannot yet be estimated; it is due solely to their liability to alcoholic neuritis, in which they amount to at least 70 per cent. The rheumatic and toxæmic forms, on the other hand, occur more frequently in males, probably from their greater exposure to the causes of the disease.

More than one cause may co-operate in producing the affection, and then we may distinguish the one as "predisposing," the other as "exciting," although, as a rule, either would be adequate alone were its degree more intense. The double causation can most often be traced when cold or some depressing influence co-operates with alcohol, or when the influence of the latter is conjoined with that of tubercle, as either the primary or secondary cause. Even more frequently the symptoms follow some cause of constitutional depression, inadequate alone. In one case this was an attack of acute gastric catarrh with vomiting, the result of the alcoholism which was the real cause of the neuritis. In other cases a depressing emotion has seemed to be the excitant, and, as we have seen, this appears to be sometimes alone sufficient. In one alcoholic case the injurious effect of painful emotion (due to the death of a son) was at once to arrest improvement, and to excite a progressive increase in the symptoms, which continued until death a few months later. Anæmia probably acts chiefly as a predisponent, lowering the nutrition and resisting power of all the tissues, and rendering a slighter cause effective. Thus, in the case of an anæmic girl recorded by Barrs, the symptoms followed immediately on bathing the feet in cold water, when the patient was heated.

Symptoms.—The symptoms produced by multiple neuritis differ widely, as has been already intimated, according to the character of the affection of the nerves. They are of three classes—motor weakness, sensory disturbance, and inco-ordination. Psychical symptoms are sometimes pronounced, especially in alcoholic cases. Loss of memory, especially as regards place and time, are the most obvious conditions, but hallucinations, especially visual, are not infrequently present. The motor weakness depends on an affection of the motor nerve-fibres, and usually involves first and chiefly the flexors of the ankle and extensors of the toes, and the extensors of the wrist and fingers in the forearm, muscles that are homologous and supplied by corresponding nerves, the anterior tibial or peroneal nerve in the leg, the radial branch of the musculo-spiral in the arm. The result is the characteristic "wrist-drop" and "foot-drop," shown in Fig. 58. Other

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* Schlesinger ('Neur. Cent.,' 1895, p. 578) describes a form of neuritis associated with *endarteritis obliterans* similar to that occurring in syphilis.

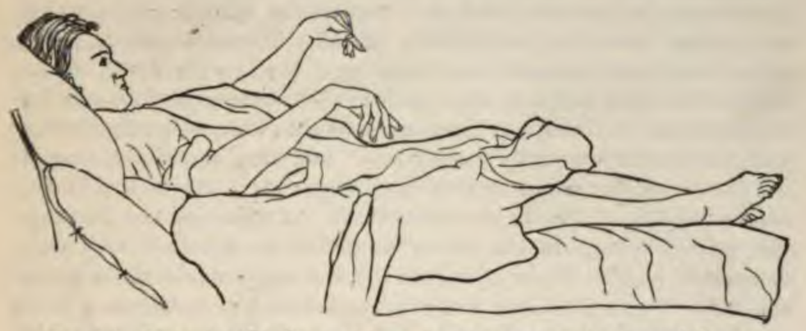

FIG. 58.—Multiple alcoholic neuritis; palsy of extensors of wrist and flexors of ankle. (From a photograph by Mr. Hyde Marriott, B.Sc.)

muscles suffer in severe cases. The sensory symptoms consist of subjective sensations of tingling, &c.; pains of various character, seat, and degree, often referred to the nerves or deep parts; tenderness of the skin, nerve-trunks, and muscles, and loss of cutaneous sensibility. The inco-ordination resembles that present in the slighter degrees of locomotor ataxy, and is often associated with loss of the muscular sense. These symptoms will be considered in detail. According to the predominance of one or other of the three sets, corresponding varieties are distinguished,—(1) a *motor* form, in which there is loss of power with or without the other symptoms; (2) a *sensory* form, in which there is no marked muscular weakness or inco-ordination, but in which sensory disturbance—irritation or loss—is the chief symptom; (3) an *ataxic* form, in which, without marked loss of power, with or without sensory symptoms, inco-ordination is the dominant symptom.

As mentioned in the introductory section, the varieties of polyneuritis that are due to special metallic poisons and to certain acute specific diseases are described separately, in the second volume of this work, among the general diseases of the nervous system. The common alcoholic form is alone suitable to convey a knowledge of the general features of the affection, but the account of its symptoms must be completed by some mention of those of the less common forms, which do not need separate description elsewhere.

It should be clearly understood at the outset that no set of symptoms is exclusively related to a single cause. The various forms which depend on the incidence of the disease on the motor or the sensory nerves in preponderant degree, are met with in consequence of each of the common causes, although, as we shall see, some causes give rise more frequently to one form than to another.

The onset and course of multiple neuritis vary much in different cases, both of the same class and of the several classes. Premonitory symptoms occur in some cases. They have been observed chiefly in the alcoholic form, but occur in others also. When due to cold or other causes of constitutional disturbance, the special symptoms of the general state may immediately precede the onset. Thus, after exposure to cold, general rheumatic symptoms, with fever, bronchitis, and other signs of catarrh, may immediately be followed by the symptoms of neuritis. The symptoms that have been regarded as premonitory are especially "numbness" and tingling of the fingers and toes, or of the palms and soles, or the lower parts of the limbs, vaso-motor disturbance in the extremities (as pallor of the fingers), and painful cramp in the calves or elsewhere, together with dull rheumatoid pains. These constitute the first symptoms of the disease, but when they exist alone for months before the onset, they may be regarded as "premonitory." All these symptoms are probably due to the influence of alcohol on the nerves, disturbing the function of the structures whose nutrition undergoes subsequently a corresponding impairment.

The onset itself may be acute or subchronic, and is sometimes so slow as to deserve the name of chronic, especially in the slighter sensory form. An acute onset is on the whole rare in alcoholic cases unless the malady is "excited" by some coöperant influence. Thus, in one alcoholic, a prostrating attack of vomiting, lasting for ten days, was followed by an onset of the symptoms of neuritis so acute that at the end of a week the patient was unable to stand, and there was general impairment of sensibility. It is met with chiefly in cases due to cold or toxæmic states; in these it may be attended by severe constitutional disturbance, rigors, and considerable pyrexia. In other forms the elevation of temperature is usually slight or moderate, but sometimes amounts to 3° or 4°, and may last from one to three or four weeks. The first definite symptom (if it has not existed before) is often the tingling, or "pins and needles" in the extremities, already mentioned, with vague "rheumatic" pains, which become more acute. Motor symptoms are usually soon added, palsy or inco-ordination, or both. They are seldom absent in acute cases. The pronounced symptoms of the disease may resemble in character those produced by the inflammation of single nerves, differing only in distribution, and involving all the functions of the affected nerves; this is often the case when the malady is acute in onset. But when chronic, as already stated, the fibres may be affected according to their specific function, and to this the symptoms are then limited, so that they resemble those that are ascribed to a disturbance of special elements of the spinal cord, rather than those of an affection of isolated nerves.

The motor form involves either the upper or lower limbs or both, but always the corresponding limbs on both sides, and their distal extremities, the hands or the feet, first and chiefly.* Whichever limbs, arms or legs, present the first symptoms, suffer in greater degree throughout, and it may be alone. The feet suffer more frequently than the hands; motor symptoms may exist in the legs, and only sensory symptoms in the hands, and this when the weakness in the legs is extreme. In whatever part the affection occurs, various sensory disturbances, and especially pains, commonly precede the weakness, and increase with it. They are accompanied by tenderness, generally along the nerve-trunks, but almost invariably in the muscles, and sometimes of the skin. These pains may occur in the limbs that do not become paralysed if the action of the cause can be arrested. Although loss of power is the obtrusive feature, some loss of co-ordination generally accompanies it, and is often the means by which the attention of the patient is first directed to the commencing disability. He finds a difficulty in balancing when standing, or in performing the finer movements with his fingers; and his attention being thus drawn to the part, he finds, on further investigation, that there is some actual defect in the power of extending the wrist and fingers, or in raising the toes or foot from the ground during the forward movement of the foot in walking. Tremor is often conspicuous in alcoholic cases long before the loss of power. Whenever there is weakness in the leg the knee-jerk can no longer be obtained, probably because the afferent muscle-nerves suffer more widely than the motor nerves. The constancy of this loss is very great, and it is not related to any one class of symptoms, but occurs equally in the paralytic and ataxic forms of neuritis. At the same time it is not absolute; the knee-jerk seems to persist in very rare cases of slight degree, as mentioned on p. 160.

When the first symptoms are in the legs, the first motor loss is the inability to raise the toes from the ground in walking, owing to imperfect flexion of the ankle-joint and extension of the toes. But this symptom may be to a large extent obscured by the interference with movement occasioned by the tenderness of the muscles and the hyperæsthesia of the skin, which commonly coexist and prevent the exertion of the amount of power that is possessed. There is a tendency for the nerves of the sole and the palm of the hand to be especially involved in the hyperæsthesia—in obedience to a law of pathological susceptibility which is to be traced not infrequently in polyneuritis, especially in the tabetic, diphtheritic, and toxæmic forms.

These effects of the sensory irritation may render it difficult to determine the common course of the motor weakness, the order in which the muscles suffer, and the relative degree of their impairment at different periods. The conspicuous fact is that the muscles first to suffer are those supplied from the external popliteal nerve (anterior tibial group), and these are followed by other muscles below the knee, except in the very rare instances in which palsy does not spread beyond the muscles first affected. The muscles above the knee suffer later and less, and more frequently escape considerable impairment. The flexors of the knee, although supplied by the sciatic nerve, do not suffer more than the extensors, or more frequently, so far as the tenderness permits us to judge. Least frequently and in least degree the muscles suffer that move the hip-joint; it is only in the most severe cases that these are appreciably affected. Very rarely is the innervation of the bladder or rectum involved in the disturbance of polyneuritis. Interference with these functions may generally be regarded as evidence that the morbid changes are not limited to the nerves, but involve the spinal cord itself.

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* The very various symptoms of the disease are well illustrated by an instructive series of cases published by Dr. Buzzard. 'Paralysis from Peripheral Neuritis,' 1886.

In the arms, the first and chief defect in power is in the extensors of the wrist and fingers. Both arms are affected together or in quick succession—one never suffers alone in considerable degree. Thus the palsy resembles at first, and sometimes throughout, that which is familiar as the effect of lead poisoning. The resemblance may extend to the escape of the extensor of the metacarpal bone of the thumb, and of the supinator longus. Lead palsy, indeed, is an example of peripheral neuritis of the "toxic form," and differs from the general form in the limitation of even severe palsy. It seems that the radial nerve (of the musculo-spiral) is that which, in the upper limbs, is the most prone to suffer—a fact which is at present unexplained. It probably depends not on any peculiarity of position, but on some deeper functional relation and position among the nerves of the body, since it obtains when the affection is of the peripheral extremities of the fibres and does not involve all the fibres of the nerve. The radial nerve is homologous with that in the leg of which the fibres are the first to suffer—the peroneal,—regarding which the same facts are true.

It is only in the cases due to metallic poisons that the palsy becomes considerable without spreading to other muscles. Generally the flexors of the wrist and fingers become weak, and so do the interossei, while the last to suffer are the thenar and hypothenar muscles. It is rare for the palsy of any muscles except the extensors to be complete. In more severe degree the muscles above the elbow suffer, last and least those of the shoulder. The muscles below the elbow may be almost powerless when those above it are scarcely affected.

In severe cases the trunk muscles are involved—the diaphragm, and the muscles of the thorax and abdomen. Less rarely, but also only in severe forms, increased frequency of the pulse and palsy of the vocal cords indicate that the fibres of the vagus are suffering. Very seldom the facial muscles, or those of the tongue, are involved. Affection of the motor ocular nerves and of the pupils has been met with only in the most severe cases, and chiefly in those due to toxæmia. Slight nystagmus is common. The nerves of the pharynx and alimentary viscera seem always to escape. It is more doubtful whether this is true of the branches to the lung.

Thus the stress of the affection falls on the muscles of the extremities, and those that are affected first also suffer alone in slight cases, and most in those that are severe. As they become weak, and sometimes even earlier, they become tender, and this muscular tenderness usually increases to an extreme degree, and becomes even more obtrusive, in many cases, than the tenderness of the nerves. The limb cannot be grasped, even gently, without cries of pain being elicited, and every change of posture gives rise to distress. This is an important sign, and is no doubt due to the fact that all the nerves of the muscle suffer, the afferent as well as the motor twigs. Very rarely it is absent. It increases the pain of an electrical examination, and, after a strong current has been applied, the muscles may ache for hours — a fact that should inculcate scrupulous consideration in all such procedures.

The affected muscles quickly become flabby and waste, and their prominences disappear. The muscular atrophy becomes extreme in some instances, but the aspect of the limbs differs from that in progressive muscular atrophy on account of the partial distribution of the affection, and because it is only in a few muscles, such as the extensors of the fingers and the interossei, that the wasting is as great as in that affection. Changes in the electrical excitability of the muscles concur with these symptoms; they present the reaction characteristic of nerve lesions, described at p. 72. The faradic irritability is lost, and that to voltaism is increased in amount, and often but not always altered in quality, in the usual manner. In slight cases, however, increase in voltaic irritability may be trifling, and may be the chief change. In the nerves the irritability to both currents lessens and is ultimately lost. If a case is examined in the very early stage, the increased irritability mentioned at p. 76 may be found, and afterwards replaced by diminution and loss. In the most severe cases the muscles most affected may quickly lose all irritability, on account of the intensity of the secondary changes in the muscular tissue, which destroys instead of merely changing their special structure and characteristic functions. Occasionally the wasting is concealed by œdema, but now and then the size of the muscles may be little reduced even when they are totally paralysed, with extinction of faradic and great reduction of voltaic irritability. But the wasting is only deferred, and extreme atrophy ultimately occurs. In such cases there is probably a rapid fatty degeneration of the muscles, and the fat accumulates between the fibres so as to maintain, for a time, the bulk of the muscle. An extremely rare alteration is the preservation of some voltaic irritability in the nerves as well as in the muscles, faradic irritability being lost in both.

As in other cases of unequal palsy, the less affected muscles of the limbs are apt to undergo contracture and shortening, especially when the palsy causes the part to assume habitually a certain position under the influence of gravitation, or a certain posture is adopted by the patient in order to lessen pain. These contractures occur chiefly in the lower limbs. The foot-drop shown in Fig. 58 is especially frequent, from the palsy of the flexors permitting the foot to fall as the patient lies in bed, under the influence of gravitation and of the unopposed extensors. The effect of posture is seen in the contraction of the flexors of the knee, which may be fixed at a right angle, and to a less degree of those of the hip, on account of habitual flexion of these joints to escape the pain occasioned when the parts are put on the stretch in the extension of the limb.

The affection of the sensory nerves of the skin is sometimes absent; more often it is conjoined with that of the motor nerves, and sometimes only with that of the sensory muscle-nerves. It very seldom exists alone. Its first manifestation is the tingling and other subjective sensations already mentioned, to which is soon added the hyperæsthesia which is often so troublesome throughout the disease. Sensory disturbance is usually more extensive than the motor palsy, but is greatest at the most affected parts, the extremities of the limbs. Except in slight cases, the damage to the sensory fibres becomes sufficient to lessen the sensibility of the skin to touch. A faint touch may be unfelt while over-sensitiveness to pain continues, and is even so intense that a strong touch may cause distress. Rarely, as I have twice seen, sensibility to pain is lost, that to touch being preserved. The temperature sense is usually normal. These sensory changes are first developed on the extremities, sometimes on the palms and soles; they may even be still more local, and only considerable on the tips of the fingers, or the hyperæsthesia may be chiefly under the nails. Spontaneous pains accompany the hyperæsthesia, dull, or acute, or "burning" in character, referred to the deeper parts of the limb, or the nerve-trunks, or the joints, and increased or excited by any attempt at movement. The pain referred to the joints may be really in them, especially in the gouty, or it may be in the nerves that pass by the joints and are disturbed mechanically by their movement. The sensory change increases in area as the disease develops, and usually extends first up the outer side of the leg and the radial side of the forearm. Although sensibility to pain is seldom lost, the conduction of pain is delayed in some cases.

In the ataxic form, inco-ordination is the chief symptom, and it may accompany the onset of muscular weakness in the motor form. It is usually accompanied by muscular tenderness, and exists with or without the affection of cutaneous sensibility just described. It probably depends on changes in the afferent muscle-nerves, the impressions from which are chiefly concerned in the process of co-ordination (see Spinal Cord, Functions). The implication of these nerves is shown by the muscular tenderness that is so marked a feature. The inco-ordination in these cases resembles closely that which exists in tabes, and the knee-jerk is lost as in that disease, while the frequent occurrence of pains in the limbs increases the resemblance. Hence the form in which this symptom is predominant has been called *pseudo-tabes*, often with the prefix *alcoholic*, from the fact that this is its most frequent cause. It has, however, also been met with from arsenical poisoning. The ataxy may involve the arms or the legs, but is most frequent in the latter. It never reaches the degree met with in advanced tabes, probably because very extensive and intense damage to the muscle-nerves only occurs when the toxic cause is intense; its effect then is not limited to a single set of nerves, but, involving the motor also, produces a loss of power, before which the inco-ordination ceases to be prominent, if it does not of necessity disappear. The general law that limitation needs chronicity of course and moderation of degree holds good in these cases on the sensory as well as on the motor side. When the inco-ordination is the prominent symptom the disturbance of sensory nerves is slight, and if there is extreme hyperæsthesia and much loss of sensation there is usually also motor palsy; and thus this ataxic form is not commonly accompanied by the intense sensory disturbance of the complete and characteristic cases. Sensory loss, chiefly to touch, is, however, occasionally met with in chronic cases of the ataxic type. The resemblance to tabes is then still greater. The distinction is considered in the section on Diagnosis.

Tenderness of the nerve-trunks in the region of whose supply the symptoms are greatest is a very common symptom, although not always readily recognised, because they are only more tender than the adjacent structures, and the degree of difference may seem not great when all the parts are extremely sensitive. It is less frequently a marked symptom than the tenderness of the muscles, and must depend (since the pain is felt at the spot) on the involvement of the nerves of the sheath. It is only in very rare cases, chiefly of irregular adventitial neuritis, that any swelling of accessible nerves can be distinguished.

Reflex action from the skin varies much in its condition. In cases with hyperæsthesia it is often increased, even when there is considerable motor palsy; the muscles that escape cause the movement, and the impression that reaches the reflex centre is doubtless increased in degree just as is that part of it which reaches the brain and is felt as increased sensation. But in severe cases, with extensive loss of power and sensation, the skin-reflex may be lost. The loss may be observed to coincide with an increase in the other symptoms, and generally to be related to loss of sensibility to touch with extensive motor palsy; it may, however, be lost when sensation is perfect. Occasionally it is lost out of proportion to the other symptoms, motor or sensory. The muscle-reflex action (myotatic irritability), as we [illegible] seen, is almost invariably lost. Although the knee-jerk has been observed to persist in rare cases (Déjerine), its persistence is quite exceptional, and probably depends on the escape of at least some of the fibres on which the action depends. Possibly, however, the knee-jerk is sometimes excessive in the early stages of the disease in consequence of an irritable state of the nerves on which it depends, similar to that which gives rise to the hyperæsthesia. I have more than once found it increased in cases of slight sensory neuritis (*e. g.* with symmetrical tingling and slight anæsthesia on the soles), and in one such case it disappeared as the disease progressed. On the other hand, it is often lost when there is scarcely any appreciable weakness of the extensors, although usually some tenderness of these muscles suggests an affection of the afferent path.

Trophic changes occur in prolonged cases—in the nails, skin, and joints, and are similar to those of ordinary neuritis; glossy skin, articular adhesions, and thickening being the most common. Bedsores are not common—a fact well established, but not altogether easy to explain. The vaso-motor nerves may present disturbance of function, and some œdema of the limbs is common, especially in cases that are due to alcohol; the blood-state or impaired function of the kidneys probably aids in its production. It occurs about the ankles, back of the foot, and very frequently about the wrist and back of the hand. In one very severe and fatal alcoholic case the legs and back were the seat of a peculiar brawny œdema, pitting very slowly, and attended with lividity of the skin in the depending parts, which became brighter in colour on pressure. In this case endocarditis developed during the course of the malady. In one recorded case œdema was present only over the nerve-trunks in the neighbourhood of joints, a place in which the movement of the limbs especially irritate the sheath-nerves of the trunks, to which the vaso-motor nerves of the part are probably related. Indeed, it is common for œdema to be present about the joints when it is not elsewhere, and this may be the explanation. Pain in the joints and even some effusion into them have been observed, especially in association with an acute onset, but it is perhaps more often gouty * than rheumatic in alcoholic cases, and its significance as to the "rheumatic" nature of the neuritis must be looked on with some suspicion. It may be occasionally a result of the disease of the nerves, since chronic changes in the joints often take place just as they do in the inflammation of single nerves. It is especially common in the finger-joints and in the wrist, but it occurs also in the shoulder and knee, less frequently in the elbow, and in the joints of the leg other than the knee. There is pain on movement and thickening about the joint, with ultimate limitation of movement. It is a troublesome effect of the disease, because it interferes with movement when power returns.

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* Acute gout, it must be remembered, sometimes attacks the larger joints with or before the smaller ones, and may closely resemble an attack of rheumatic fever.

There is much more tendency to it in some cases than in others, and it may be so extensive as permanently to cripple the sufferer.* The adhesions commonly re-form after they have been forcibly broken down.

The symptoms are wide-spread in proportion to the acuteness and intensity of the malady, but differ in these respects also according to its cause. The effect of metallic poisons is usually limited and confined to the arms, as in lead palsy. In alcoholism all the limbs are often affected, but the arms escape more often than the legs. From malaria the legs usually suffer alone; such cases are considered at p. 185. In cases with complex causation a general parenchymatous neuritis may be accompanied by a preponderant adventitial inflammation of some one nerve, as the facial or sciatic. The cases with most widely distributed symptoms are those that result from obscure toxæmic states, from cold, and from alcoholism. It is in these, and especially in the first, that the nerves of the heart, larynx, and of the muscles of respiration most frequently suffer. When the laryngeal nerves suffer it is usual for both adduction and abduction of the vocal cords to be weakened, seldom one movement only. The optic nerves have been affected only in rare cases with considerable blood-change. In the case mentioned on p. 160 (in which there was brawny œdema and endocarditis) there was slight optic neuritis and a diffuse œdematous retinitis.† The face has also been affected on both sides in some of the cases due to exposure to cold, and also, usually in slight degree, in some toxæmic cases; it seems always to escape in cases due simply to alcohol. The sphincters almost always escape; a slight impairment of function has indeed been met with in a few very severe cases of probably pure polyneuritis, but their affection, as already stated, usually indicates an implication of the spinal cord.

The *Course* of the affection is determined by the same conditions as influence its intensity, and by the extent to which the cause is under control. The more severe and acute the symptoms, the wider is their range and the longer is their duration. Very commonly the symptoms increase during three to six weeks, then become stationary (if the cause is stopped), and after one or two months slowly improve. The first sign of improvement is a diminution in the pain and hyperæsthesia, but the tenderness of the muscles and the nerve-pains on movement often continue long after the spontaneous pains have ceased. If tingling, &c., ceased as the disease advanced, their return may herald improvement. Power slowly returns, first in the muscles affected last and least, and afterwards in those paralysed and wasted in greater degree. In these the weakness lasts for many months, and shortening of the opponents of the most affected muscles is apt to occur. The contracture of the calf muscles, secondary to palsy of the flexors of the ankle, constitutes a grave hindrance to the use of the legs in standing and walking. The patient cannot get the sole and heel on the ground so as to afford a uniform base of support, and keep his body upright so as to balance it. But this contracture invariably yields to persistent efforts to walk. The palsy of the arms lasts longer than that of the legs, or *vice versâ*, according as the one or other suffers most. The excess of impairment of the muscles most affected is often very conspicuous during recovery. Improvement goes on for a long time, generally until recovery is perfect, but very rarely there is some permanent loss of power. It is remarkable, however, how almost complete a recovery may take place, even after the paralysis has been considerable for a year. The wasting lessens and power returns; latest where there is most loss of irritability, and usually later in the small muscles of the hand than in the muscles of the forearm. Relapses are apt to occur, but only when the cause of the neuritis is still in operation in some degree, or when the patient is again exposed to it or to some adjuvant cause, such as cold.

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* As in a case recorded by Taylor, 'Guy's Hosp. Rep.,' 1888.

† The changes are described by Edmunds and Lawford, 'Trans. Oph. Soc.,' ix, 137.

But the malady sometimes runs a very acute course, especially in the cases due to toxæmia or to cold, so that all parts of the limbs have become weak and the extremities powerless by the end of seven or ten days, and the involvement of the nerves of the respiratory muscles may bring life to an end within a fortnight, sometimes even within a week. In this form there may be severe initial pain, not specially related to the regions afterwards affected. In one case it was severe in the back, in another across the abdomen. It is probably a direct effect of the blood-state, distinct from its influence on the peripheral nerves. Such cases bear considerable resemblance to those of "acute ascending paralysis" in their course—differing, however, in the fact that the ascension is up the limbs rather than up the general frame. Sometimes, on the other hand, the disease presents a course far more chronic than that described above, and occupies many months in its development and progress. This is often the case with the limited alcoholic forms, especially the ataxic variety, "pseudo-tabes," and with cases in which the toxic agent is absorbed from without very slowly, and its effect accumulates gradually, as in some cases of chronic arsenical poisoning. Slight cases, again, treated promptly have been known to recover in a month.

The temperature, usually raised in an acute onset (see p. 154), may be normal in chronic cases, with or without an occasional rise, or may be slightly raised during the chief part of the course of the disease. A persistent considerable elevation suggests some complication, such as phthisis, and should lead to careful and repeated examination of the lungs. It may also indicate a persistence of toxæmia, other indications of which are mentioned among the complications of the disease.

The Cause of Death in acute cases is often palsy of the respiratory muscles, sometimes aided by a catarrhal bronchitis, or paralysis of the heart when the vagus is affected; in more chronic cases it may be caused by exhaustion, due to chronic gastric derangement and the wearying effects of prolonged suffering, or the effects of disease of the spinal cord. Pneumonia frequently causes death in the acute forms, and in alcoholic subjects the inflammation of the lung so often runs a low course, with extensive consolidation, sometimes bilateral and unusual in seat (as in the middle lobe and central portions of the lung), that it is difficult to avoid the suspicion that its form and course are determined by coincident neuritis of the pulmonary branches of the vagus, either primary or secondary to the inflammation of the lung. Death may result unexpectedly from cardiac failure when no preceding symptoms have suggested an affection of the cardiac nerves, and in such cases there is generally a coincident degeneration of the walls of the heart. Fatal neuritis of the vagus, however, may occur very early in relapses. Other coincident effects of chronic alcoholism are frequent causes of a fatal issue, such as cirrhosis of the liver, gastric troubles, and chronic cerebral meningitis in cases of alcoholism, or acetonæmia in cases of diabetic polyneuritis. Various morbid blood-states, causing the neuritis, may continue and cause death. Lastly, phthisis is a frequent cause of death, and probably varies in the relation it bears to the neuritis, sometimes being its consequence, and sometimes apparently a coincident effect of some obscure cause.

COMPLICATIONS.—The chief of these have been just enumerated in the list of the causes of death. The most important in alcoholic cases are the effects of alcohol on various organs and tissues, especially on the liver, heart, and spinal cord. It is very common to find the liver enlarged, sometimes very large; less commonly it is contracted. The enlarged liver may be either fatty or fibroid. Ascites and other effects of portal obstruction are much less frequent complications than might be expected, perhaps because it is rare to have much contraction of the new tissue in the liver. The kidneys also are often diseased. Gastric catarrh with its varied symptoms, morning vomiting, anorexia, and the like, is very common. So also are the several effects of gout due to the alcoholism or to metallic poisons when these have been the cause of the neuritis (as lead, or, as in one case under my notice, silver). On the part of the nervous system there may be various subacute and chronic forms of inflammation of the substance of the spinal cord or of its membranes, the common manifestations of which are to a large extent prevented by the affection of the nerves. The most important, the trunk girdle-pains and the affection of the sphincters, are further considered in the section on Diagnosis. Chronic cerebral meningitis is also common, and usually fails to produce its common manifestation, pain, even when marked and extensive opacity of the membranes and increase of fluid are found after death. It usually causes some chronic mental disturbance, often slight optic neuritis, and sometimes general convulsions. But mental change is also common as a direct effect of the alcohol on the brain; distinct delirium tremens often coincides with the onset of the neuritis. In most alcoholic cases, indeed, the mental state is unnatural; the patient is irritable, intolerant of pain, and deficient in self-control. Various manifestations of hysteria occur in women, and a peculiar childish jocularity is sufficiently common to deserve special mention. The craving for withheld stimulants increases the irritability, and, in the case of women, previous training in duplicity in order to obtain alcohol has usually induced a general deterioration of the moral sense, which has results that startle those who have been successfully deceived. Mental change is for the most part absent in the cases due to cold (save in some in which there is an intense blood-change), and in those produced by metallic poisoning; but it occurs frequently in toxæmic cases, generally in the form of simple delirium, and is apparently due to a simultaneous action of the morbid agent on the cells of the brain. A persistent morbid blood-state may cause endocarditis and other consequences.

Phthisis is also a very frequent complication; its double relation to the neuritis has been already mentioned. The form of lung disease met with in neuritis presents no special peculiarities, except that low pneumonic changes play a considerable part in the morbid process. Acute pneumonia is also common, and in the cases in which it does occur almost always fatal.

Pathological Anatomy.—The changes in the nerves correspond to those already described in the account of neuritis, with the exception that the chief changes of isolated neuritis are in the connective tissue, and especially in the sheath; and those in the nerve-fibres are subordinate. In bilateral neuritis the connective tissue presents very trifling changes. Some variations are met with in this respect; when the distribution of the affection is not strictly symmetrical, the sheath and interstitial tissue suffer in greater degree; in proportion to the acuteness and symmetry of the symptoms, the changes are restricted to the nerve-elements, and the connective tissue is unchanged, but in some cases both the connective tissue and nerve-fibres are involved, as in Fig. 59. This is also the condition when there is acute inflammation spreading from the one constituent to the other, as in the case of fatal septicæmic neuritis recorded by Roth (see p. 148). In the vast majority the nerve-elements are affected chiefly or even exclusively; the changes are essentially "parenchymatous." It is remarkable, indeed, how slight is the affection of the connective tissue and sheath, even in nerve-trunks that have been extremely tender. Hence it is probable that this tenderness is often due to changes in the nerves of the sheath, the "nervi nervorum," similar to that which the proper conducting fibres of the nerve undergo, rather than to an inflammation of the connective tissue itself.

Naked-eye changes are present only when the connective tissue and sheath are inflamed in considerable degree. In recent and acute cases of this character the nerves may be found reddened, swollen, and sometimes small hæmorrhages are visible. In older cases they may or may not be swollen, but are usually softened, and even pulpy. When the changes are confined to the nerve-fibres, diminished consistence may be the only alteration that can be recognised on simple examination; and often this is absent, and the nerves appear normal on external examination, even when their fibres are extensively disintegrated. Sometimes under a low magnifying power a section of the nerve has a mottled aspect, due to the irregular destruction of the fibres.

When the connective tissue is involved the microscope shows the sheath to be infiltrated with lymphoid cells, and in old cases many spindle-cells and fibres increase the bulk. The same changes may be traced in the septa between the fasciculi, and in the secondary sheaths that surround the latter. The walls of the vessels are also thickened (Fig. 59, A). When mere acuteness of parenchymatous neuritis causes the connective tissue to be inflamed, the interstitial tissue and inner part of the sheath are chiefly affected, and an amorphous "exudation" may be found in these situations. But the fibres themselves always suffer in conspicuous degree; and in the majority, as in the case from which Figs. 59 and 60 are drawn, they suffer chiefly, the interstitial tissue presenting comparatively little change. In this case the changes were not purely toxic; there was a complex state, combining inflammation of the fibrous elements of the nerves with the purely parenchymatous change. The patient was a woman aged thirty-three, alcoholic; the course of the disease was subacute; the symptoms reached a considerable degree in a few weeks, and the patient died, chiefly from coincident liver disease, five months after the onset. The symptoms were characteristic, and continued up to the time of death. The fibres present the alterations met with in all acute forms of neuritis,

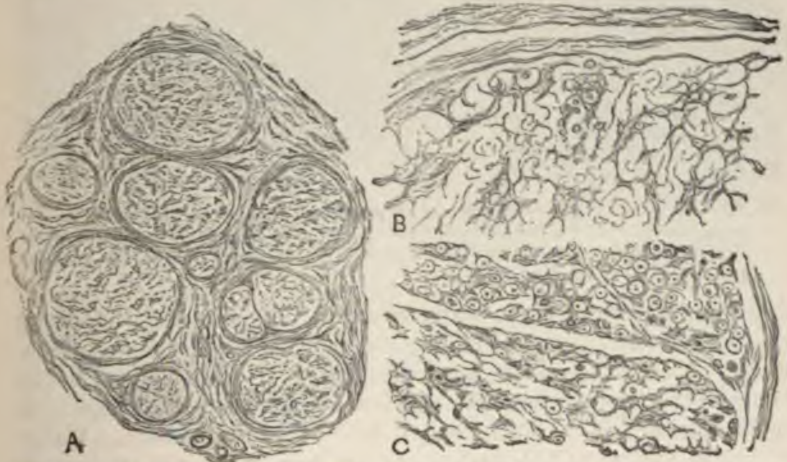

FIG. 59.—Multiple alcoholic neuritis: sections stained with carmine and cleared. A. Transverse section of part of sciatic nerve; low power. B. Part of a fasciculus of same, more highly magnified. C. Part of a less affected fasciculus from a musculo-spiral nerve.

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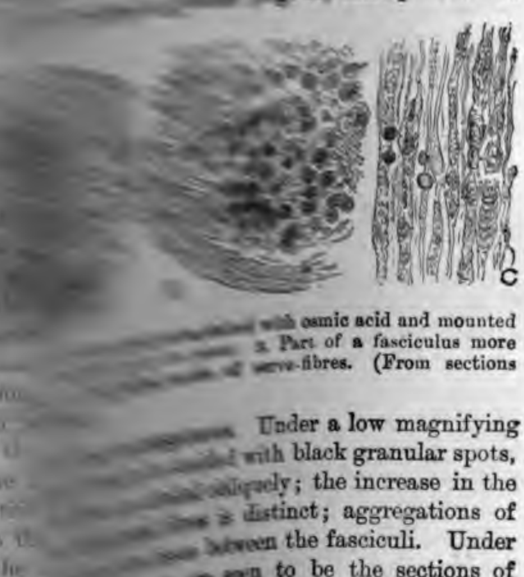
occur almost [illegible]



[illegible] with osmic acid and mounted [illegible] Part of a fasciculus more [illegible] nerve-fibres. (From sections

PATHOLOGICAL [illegible]
those already [illegible]
that the chief [illegible]
and especially in the [illegible]
dinate. In bilateral [illegible]
changes. Some [illegible]
tribution of the [illegible]
interstitial tissue [illegible] Under a low magnifying
ness and symmetry [illegible] with black granular spots,
nerve-elements, and [illegible]; the increase in the
cases both the [illegible] distinct; aggregations of
Fig. 59. This is also [illegible] between the fasciculi. Under
tion spreading from [illegible] seen to be the sections of
of fatal septicæmic [illegible] two or more being blended
majority the [illegible] section (C) the process of
the changes are [illegible] seen, and from it the
indeed, how slight is the [illegible] be better understood. The
even in nerve-trunks [illegible] the fibres at some places,
probable that this [illegible] the sheath is left empty.
the sheath, the "nervi [illegible] outside the fibres. The
conducting fibres of the [illegible] considerable size, is seen in
of the connective tissue [illegible] fibres and cells lie between

Naked-eye changes are [illegible] are round and spindle cells
sheath are inflamed in [illegible] which they have taken
of this character the nerves [illegible]
sometimes small hæmorrhages [illegible] the nerves varies much in
may not be swollen, but are [illegible] of the sheath may exist

greater in one region than elsewhere, and especially marked in the posterior columns. Such alterations are most marked in chronic cases in which the symptoms have continued for months, and especially in those due to chronic alcoholism, or in which life has been prolonged in a state of disability.

Other organs are often found diseased, and in alcoholism the lesions common in that disease are generally found in some degree, especially alterations in the liver and kidneys. The former may be large and either cirrhosed or fatty; the kidneys are generally large and opaque, sometimes with fatty striæ in the cortex. Pneumonia is frequent both in these and in all cases due to causes that induce an intense change in the blood. In these there may also be enlargement of the spleen, such as is met with in typhoid fever and septicæmia. Phthisical changes in the lungs are also common in chronic cases, both in those that are due to alcohol and in others. No special peculiarity has been observed in the pulmonary lesion, but it is very desirable that more attention should be paid to the state of the nerves of the lungs entering the diseased parts. When not otherwise diseased, the lungs are generally found to be the seat of terminal congestion and œdema. The walls of the heart may be degenerated, and alterations may sometimes be found in its nerves.

Pathology.—An outline of the chief facts in the pathology of multiple neuritis has been given in the introductory section, and not much remains to be added. The relation of the motor symptoms to the lesion of the nerves has also been described in the general account of diseases of the nerves, and in the description of isolated neuritis; all that has been said of the single form is true also of that which is multiple. The wasting that accompanies the loss of power, and the change in electrical irritability, are the same in each. In each it is true, moreover, that the position of the lesion in the course of the nerve-fibre makes no difference to the symptoms; these are the same whether the disease is in the trunk of the nerve, the peripheral fibres, or the actual nerve-endings. The relation of the ataxy in the pseudo-tabetic form to a neuritis of the afferent muscular nerves is proved partly by exclusion, partly by analogy, since in true tabes the lesion may be confined to these; the spinal cord may be free, and only the peripheral nerves diseased. The evidence for this will be described in the general account of the functions and symptoms of diseases of the spinal cord, and in the chapter on locomotor ataxy—a disease which sometimes not only resembles, but is almost identical with, the pseudo-tabetic form of neuritis. The pains and hyperæsthesia are explained by the fact that the disease of the nerves is one in which the nerve-fibres themselves suffer primarily, and that on them the cause must have a primary and direct action.[*] An influence which leads to the molecular destruction of the nerve-fibres must, in destroying their structure, disturb gravely their function; and this disturbance of structure and of function proceeds in the parts still connected with the nerve-centres, so that the irritative influence of the molecular changes can act on the centres by the normal upper parts of the nerves throughout the whole course of the malady. It induces in the centres, as all prolonged pain does, an undue readiness of action, a state of over-excitability; and so we can understand the persistence of the pains and hyperæsthesia, and the fact that these continue until the process of restoration has become definitely established. The same considerations apply to the tenderness of the muscles, since the afferent muscular nerves correspond in the direction of their function to the sensory nerves of the skin. In both cases mechanical influences constitute the chief mode of their normal stimulation, and hence they may well be morbidly sensitive to such influences. The peripheral distribution of the changes indicates, moreover, that probably the first to suffer are those in which functional susceptibility is most highly developed—the actual endings of the nerves, and the structures which are especially adapted for the reception of stimuli and the production of nerve-force.

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[*] In the first edition I suggested that the degeneration of some of the fibres might be secondary to their damage by the inflammation of the sheath and interstitial tissue at some higher spot. But this now seems very unlikely; it is far

We have seen that two facts give us the key to the pathology of the disease so far as this is yet understood. One is its symmetry; the other is the fact that it is commonly a disease of nerve-fibres—that these, and not the connective tissue, are the parts primarily and specially diseased. In all structures, in proportion to the intensity and acuteness of the parenchymatous affection, the interstitial tissue participates in the changes, and escapes in proportion to its slowness and slightness. The extent to which this is true of multiple neuritis has been already pointed out. Further, in its relation to a morbid blood-state as its sole cause, we have the key to the symmetry of the affection as well as to its parenchymatous nature, and on this point there remains little to add to what has been already said. Such a limitation to the nerve-fibres as is seen in multiple neuritis is unknown in the isolated form, in which, as in gouty or syphilitic neuritis, the affection is chiefly one of the sheath and interstitial connective tissue; and the fibres suffer only secondarily in chronic cases, although, in accordance with the law already stated, they are involved in acute cases in proportion to the intensity of the process.

It is not surprising that the symptoms of polyneuritis should bear so close a resemblance to those of some affections of the spinal cord, since we must regard the peripheral nerves, at least the motor fibres, as essentially outlying parts of the cord, and it will be seen that one central disease, primary lateral sclerosis, presents many points of correspondence with the chronic form of polyneuritis, being

more probable that all the fibres suffer in the same way, and the more thoroughly cases are studied the more clearly does it appear that all symmetrical neuritis is peripheral and parenchymatous.

an affection of the fibres proceeding from the motor cells of the brain analogous to that of the fibres proceeding from the motor cells of the cord. We can understand the peripheral distribution of the disease by the fact already pointed out—that the nerve-fibres are really the prolonged processes of the nerve-cells, sharing the life of the cell, and depending on it for their vitality. The influence on which their life depends must lessen with the distance from its source, and hence their vital power of resisting morbid agencies is least at the periphery. The conditions of life of the sensory fibres, it need hardly be pointed out, differ little from those of the motor, since the ganglia containing their cells are near the cord, and almost as far from the periphery as are the motor nerve-cells. This fact also has a relation to the parenchymatous nature of the disease, since we can find no reason why the connective tissue of the nerves should suffer most at the extreme periphery. On the contrary, the inflammations that begin in the sheath are generally situated in the course of the nerves, at some spot exposed to the influence of an exciting cause such as pressure or motion. The same fact (that the cause is a blood-state) affords also an explanation of many facts of distribution. In addition to the points already mentioned it is only necessary to point out how common are instances of selective action of toxic agents upon various parts of the nervous system; and analogy prevents us from feeling any difficulty in at least accepting the fact that a blood-state should act upon one set of nerve-fibres rather than upon another, or on the fibres for the extensors, or even first and chiefly on only some of the nerve-fibres of a certain set of muscles. We do not yet know what are the influences that determine such susceptibility, but it is probable that a very slight difference in nature is sufficient to determine a very great difference in the result. The fact of symmetry excludes, as we have seen, any other causal influences than those which act through the blood, or consist in a vital tendency, and act on all structures with similar vital conditions. We know nothing as yet of the precise character of the morbid influence which causes the malady, beyond the facts mentioned in the introductory section. The greatest difficulty is presented by the fact that a poison generated through exposure to cold, or possibly by a growth of organisms in the body, should have an influence similar to that which is exerted by metals or by alcohol. At the same time it is instructive to note that the most recent researches in bacteriology point to the production of chemical substances by the disease-causing organisms in the course of their growth, and suggest that these chemical products, rather than the organisms themselves, act upon the nerves.* Alcohol, it may be further noted, is itself the result of the growth of organisms almost as low in the scale as those which are the causes of disease; the process of the growth of bacteria in the body may be not unlike a process of fermentation, and the results of the one not unlike those of the other. The analogy is the more pertinent, since multiple neuritis molecular destruction of the nerve-fibres must, in destroying their structure, disturb gravely their function; and this disturbance of structure and of function proceeds in the parts still connected with the nerve-centres, so that the irritative influence of the molecular changes can act on the centres by the normal upper parts of the nerves throughout the whole course of the malady. It induces in the centres, as all prolonged pain does, an undue readiness of action, a state of over-excitability; and so we can understand the persistence of the pains and hyperæsthesia, and the fact that these continue until the process of restoration has become definitely established. The same considerations apply to the tenderness of the muscles, since the afferent muscular nerves correspond in the direction of their function to the sensory nerves of the skin. In both cases mechanical influences constitute the chief mode of their normal stimulation, and hence they may well be morbidly sensitive to such influences. The peripheral distribution of the changes indicates, moreover, that probably the first to suffer are those in which functional susceptibility is most highly developed—the actual endings of the nerves, and the structures which are especially adapted for the reception of stimuli and the production of nerve-force.

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In each disease there may be a febrile onset, muscular wasting with the reaction of degeneration, initial rheumatic pains, and a tendency to the spontaneous recovery of the least affected parts. The distinction rests on the symmetrical localisation of the neuritic palsy, while that of polio-myelitis is characteristically random in distribution; on the persistence and severity of the neuritic pains; on the tenderness of the inflamed nerve-trunks; and on the changes in sensibility, a symptom never present in polio-myelitis. The electrical reactions are the same in both affections. It has been said the early extinction of voltaic irritability in the muscle (see p. 72) is met with only in disease of the nerves, but this is incorrect; I have more than once met with it in central affections. But we have seen that it is probable that the nerve-trunks are sometimes inflamed in polio-myelitis, and suffer as a simultaneous effect of the cause of the spinal lesion. In this condition, however, the spinal symptoms preponderate, and the distribution of the neuritis is irregular. In all cases the presence of increased myotatic irritability, or of the spasm in the legs that accompanies considerable excess of this, may be taken as proof of disease of the cord; all active muscular spasm, indeed, has this significance. Thus if the arms are paralysed, even though the paralysis closely resembles that of polyneuritis, and the legs are weak, with excessive knee-jerk and foot-clonus, the disease is of the cord, and not of the nerves. The greatest difficulty is presented by some sensory cases in which there are only tingling and numbness in the legs, and also by the problem of detecting cord disease in the presence of peripheral neuritis. The latter will be considered further on. The former is generally decided by the presence or absence of the knee-jerk, and by the fact that in peripheral neuritis the sensory symptoms are more peculiar and symmetrical than in cord disease; there is tingling or anæsthesia, for instance, in the palms or soles, or both, and a careful examination of the nerves and muscles will generally reveal a condition that is characteristic.

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* A theory apparently first suggested by Rosenheim, 'Arch. f. Psych.,' xviii, 3.

Pachymeningitis, damaging the nerve-roots, may cause paralysis, wasting, and anæsthesia, but in this all four limbs are not affected; the legs rarely suffer; the anæsthesia invades the upper parts of the limbs or the trunk as much or more than the distal parts; there is no tenderness of nerve-trunks, and there is usually distinct evidence of damage to the spinal cord itself. The mysterious disease, "acute ascending paralysis," may resemble the most rapid form of multiple neuritis, but in it the symptoms ascend the trunk from the legs to the arms, and do not begin in the hands and feet at the same time or successively, and involve the trunk last, as does the usual form of multiple neuritis. Many cases, perhaps indeed most, are of toxic origin, and are really neuritis. Diphtheritic paralysis is of uncertain nature, but there is no considerable pain, and the weakness in the limbs usually succeeds paralysis of the palate and ciliary muscle, never met with in other forms of polyneuritis. The distinction from other diseases (as, for instance, that from the symmetrical paralysis of the extensors which may occur at the onset of progressive muscular atrophy) will be considered when these diseases are described.

The distinction of polyneuritis in its ataxic form, "pseudo-tabes," from true tabes is often beset with great difficulty. This is indeed natural, since the lesion in true tabes may be identical with that in the neuritic affection; not only is degeneration of the peripheral nerves a common lesion in tabes, having the same distribution at the peripheral extremities of the nerves as in polyneuritis, but in the variety termed "neuro-tabes" the lesion consists only in this nerve degeneration, and the spinal cord is free from disease. We have, then, affections identical in pathological anatomy, and almost identical in symptoms. Yet we must class the neuro-tabes with the spinal form, from which polyneuritis, as here described, has to be distinguished; it is necessary to do so on account of the relations and etiology of the two maladies as well as on account of practical considerations. The presence of actual paralysis, of distinct weakness of the extensors, in addition to inco-ordination, would of course decide the question, since there is no actual loss of power in tabes: hence a distinction in gait pointed out by Westphal and Charcot, that in pseudo-tabes the feet are raised too high on account of the difficulty of getting the toes off the ground (like a person stepping over low objects), is of little actual value; it only exists when there is distinct loss of power. But it is in the cases of neuritis in which there are only sensory symptoms that the difficulty especially exists. It occurs, moreover, chiefly when the ataxy is moderate in degree; when this is so great that the patient is not able to stand alone, in spite of the power of moving all joints of the legs with fair force, true tabes should alone be thought of. The cases of tabes in which there is moderate inco-ordination of movement, distinct unsteadiness on walking, whether increased by closure of the eyes or not, and distinct irregularity and uncertainty in the movement of the feet and legs, are those which are perfectly simulated by the pseudo-tabetic form of polyneuritis. Pains are common to the two diseases, and neither prolonged acute pain nor dull rheumatoid pains afford the means of distinction. True "lightning" pains are seldom met with in neuritis; they are therefore strong evidence that the case is one of tabes. Extreme hyperæsthesia is much more common in neuritis than in tabes, but is of little service in the diagnosis because it is not often present in ataxic polyneuritis, but the muscular tenderness of neuritis is of considerable diagnostic value. A great excess of the reflex action from the skin is occasionally met with in tabes, the sensory impressions being painless; while in ataxic neuritis the reflex action is seldom excessive, and is generally diminished, while the sensory impression is commonly painful from the hyperæsthesia.

Some other symptoms are also of value as indications of tabes rather than neuritis,—an affection of the sphincters; a sense of constriction round the trunk (which may be felt over a wide area as a band rather than as a cord); and lastly (but among the first in importance), the reflex action of the pupil to light is often lost – seldom if ever in neuritis. Since it may be normal in tabes, its loss is of more significance than its presence, but the cases of tabes in which it is preserved form only a small minority, so that its preservation is often at least corroborative evidence that the case is one of polyneuritis. Mental change is of little value in the diagnosis unless it is in the form of definite delirium; but the less common symptoms of tabes, if present, often afford proof of the nature of the malady, although generally in association with other symptoms rather than by themselves. Optic nerve atrophy, for instance, is almost unknown in the neuritic "pseudo-tabes," and so also are the various "crises:" care must be taken not to confound the vomiting of alcoholic gastric disturbance with the gastric crises of tabes. The trophic changes differ in the two: the tabetic enlargement of the bones and disease of the joints are unknown in neuritis; and so, in the former affection, are the simple arthritic adhesions and the "glossy skin" of the latter. If all the symptoms are considered separately for their individual value, and conjointly for their associated significance, it is not often that the observer will remain in doubt.

One other special diagnosis should be mentioned, that for hysterical palsy. This occurs chiefly in women when the symptoms are motor and are in the legs. When in the arms, the characteristic situation of the palsy usually attracts attention and prevents error. A defect in the power of extension of the wrist has probably never been met with as a consequence of hysteria. Slight ataxy may, however, be purely functional in nature, but the knee-jerk is not lost, and if there is anæsthesia it is much more extensive in the functional affection.

It is of great importance to know whether the spinal cord is suffering as well as the nerves. The following symptoms are those that are the most common and important evidence of this complication:—(1) Any impairment of the functions of the bladder or rectum—incontinence of fæces, retention or incontinence of urine. It is not, indeed, improbable that their impairment may form part of the most intense form of polyneuritis, but it is not part of the ordinary phenomena of the disease; hence, in such cases, these symptoms suggest that the disease involves the cord as well as the nerves. It is important to remember that when there is mental change, evacuations into the bed are frequently without significance as to the state of the sphincters, and are due solely to the inertia of the mind. This is a fertile source of error. (2) A distinct sense of constriction, "girdle-pain," referred to any part of the trunk, probably always constitutes proof of damage to the cord. Of the same significance is sharp pain radiating around the trunk at a definite level and not elsewhere. (3) An important distinction in alcoholic cases is an unusual distribution of the affection, *e. g.* complete paralysis of all parts of the legs, or weakness of the muscles above the knee, and not of those below. Impairment of sensation up to a certain level on the legs or trunk has the same significance; disease of the nerves does not involve those of sensation up to a definite level. Bedsores also increase the probability that the cord is affected.

If a case is recognised as one of multiple neuritis there remains the question, what is its cause? In many cases this question does not arise, because the cause is obtrusive, and has been recognised as one of the elements of the diagnosis; but in other cases the cause is not clear, the diagnosis has been made independently of it, and it remains to be discovered. For the most part it is only necessary to know the various causes in order to determine which is effective in the patient under observation, because one or another of them is to be discovered without difficulty, and the rest are absent. It may be useful, however, to remember that, as a rule, in chronic metallic poisoning the arms suffer before the legs, and that in lead poisoning the acute paralysis is limited to the arms; in arsenical poisoning the affection of the arms is soon followed by that of the legs, while in alcoholic cases the legs generally suffer first. In a case in which arms and legs are affected simultaneously metallic poisoning would be a very unlikely cause. An initial affection of the proximal parts of the limbs at the same time as the distal parts suggests some other cause than alcohol, especially cold or toxæmia. Early affection of the muscles of the trunk or of the bulbar nerves has the same significance, and so also has severe constitutional disturbance at the outset. It must be also remembered, however, that alcoholism often co-operates with other causes, especially with cold; and it must also be remembered that the inquiry needs to be exhaustive before the influence of alcohol can be excluded. This is especially true in the case of females, who often obstinately deny that which would bring shame upon them. Exposure to cold has generally been severe; and if it has not, the co-operation of alcohol is extremely probable. Neuralgic pains of irregular distribution in addition to the symptoms of polyneuritis should suggest diabetes as a cause, but the examination of the urine for sugar should never be omitted. When polyneuritis is due to septicæmia this cause may be suggested by the fact that the patient is under treatment for some surgical ailment; while in all cases of obscure origin inquiry should be made for any acute specific disease to which the neuritis is known to be secondary, and the fact should be remembered that a considerable interval may separate the two. Some form of toxæmia is always suggested by the fact that the onset of the neuritis is associated with such pyrexia and constitutional disturbance as indicate an acute morbid blood-state. But this diagnosis often rests chiefly on the exclusion of other causes; if these are absent, the fact that neuritis is known to result from obscure blood-poisons may be allowed weight, especially if the symptoms begin in the legs or in all parts of the limbs simultaneously, and do not affect exclusively at first the extensors of the extremities. Sensory symptoms in these cases at the early stage often take the form of tingling and "numbness," and the pains so characteristic of the alcoholic form are frequently absent, or are represented only by dull aching. In slight cases of this type sensations of numbness and "pins and needles" in the legs, with readiness of fatigue and loss of the knee-jerk, may be the only symptoms. The facts that the nature of toxæmic causes is to a large extent unknown, and that the poison may probably be acquired from without, make it necessary to be prepared to find this cause of multiple neuritis under almost any circumstances. Residence abroad should suggest an inquiry for exposure to malarial influences. The possible significance of tubercle as a cause should lead to a careful examination of the lungs; but the possibility that phthisis may be an effect, and not a cause, of multiple neuritis must be borne in mind.

PROGNOSIS.—The danger to life is in proportion to the acuteness of the malady, to its severity as measured by the extent of the paralysis, and to the degree in which the strength of the patient is impaired by other maladies, or by the cause of the neuritis. It is important also to remember that when the affection has increased up to the time that the patient comes under treatment, it generally does not respond immediately to the arrest of its cause, such as the withdrawal of alcohol, but continues to increase for two or three weeks. It is this temporary progressive tendency that constitutes the graver element in the disease, and must be taken into account in every initial prognosis. Hence the forecast must be guarded in all cases that are severe when the patient comes under treatment, and most so if there has been a rapid increase in symptoms that had existed for a long time in slight degree. If the extensors of hands and feet are powerless, and the muscles connecting the limbs with the trunk are distinctly enfeebled, unless the disease has clearly begun to lessen, there is danger that a further extension may involve the muscles of respiration; this is especially great when the muscles moving the shoulder are considerably weakened. It obviously follows that the danger is still greater if the muscles of the trunk, and especially of the chest, have already given evidence of changes in their nerves. Pain in the trunk of the same character as that in the limbs is also a grave symptom if the motor power of the limbs has become small, or the case is one unusually general in its distribution, because where the sensory fibres are suffering the motor fibres are in danger also. Paralysis of the diaphragm adds considerably to the danger, and its power should be carefully watched in the manner and with the precautions mentioned in the account of its paralysis (p. 33). It may become paralysed without the fact coming to the consciousness of a patient lying quietly in bed, but a slight weakness of the intercostals will then cause considerable difficulty of breathing and an accumulation of mucus in the lungs, a condition predisposing to definite bronchitis, by which the patient may be quickly suffocated.

The importance of this point is great, since respiratory palsy is one of the most common immediate causes of death. It is probable also that deficient breathing power is one of the causes, perhaps even the chief cause, that lead to the development of the chronic phthisical lung disease that comes on in so many prolonged cases. Especially important, also, is any indication that the nerves of the heart are suffering. In an acute increase due to continued use of alcohol the vagus sometimes suffers severely before other nerves, and increasing frequency of the pulse is then of most serious significance. The prognosis should be guided by the motor rather than by the sensory symptoms. The latter may improve while the former increase without lessening the gravity of the latter indication. Persistent œdema is of bad omen, and so also are other indications of considerable blood-change.

When once the malady has become stationary, and its cause has definitely ceased, the danger to life is small, except in severe cases, in which the pains are intense, the patient helpless, or the heart is feeble, and the pulse persistently frequent, or there are signs of lung disease. In these, as in all cases, moreover, the danger is much increased by any indication that the spinal cord is involved. The tendency to recovery is far less in the cord than in the nerves; and retention of urine (with all its secondary consequences), bedsores, &c., are prone to occur, and to involve their own danger even if the disease in the cord does not increase. The prognosis as regards life in cases of moderate severity depends, indeed, as much on complications as on the state of the nerves. Signs of chronic cerebral meningitis, of degeneration of the walls of the heart, of disease of the liver and of the kidneys, also lessen the prospect of escape with life.

The danger is, however, greatest, and the mortality highest, in the acute cases that are due to some toxæmic state or to cold, and it must be estimated by the rapidity with which the paralysis develops and spreads. Whenever it becomes considerable in a few days, the danger of early extension to the mechanism for respiration is very great. In these cases, also, pneumonia frequently occurs, concomitant with the early symptoms, and few cases thus complicated recover.

In cases that are not fatal the full development of the symptoms is followed by a stationary period, which lasts for one or two months before improvement begins, and for this the patient or friends should be prepared. The course of restoration is always extremely slow; the muscles least affected regain power in from two to four months, but it is usually six months or more from the onset of improvement before the extensors are fairly strong, if their palsy was considerable or complete. The intrinsic muscles of the hand are still longer in recovery. The improvement in the nutrition of the muscles follows that in their power, so that they remain smaller than normal, long after they have regained strength. It may, indeed, be years before the muscles whose size can be well seen, such as the abductor indicis, are of normal size.

It is seldom that any permanent weakness is left unless the spinal cord is diseased, and when a patient is distinctly improving a good ultimate prognosis may be given, provided there are no complications to interfere with it. In no case with distinct spinal symptoms can we feel confident that restoration of power will be complete; and in cases in which there are indications of considerable damage to the cord it is almost certain that some lasting weakness will be left.

The electric irritability of the muscles and nerves may be expected to return to the normal more slowly than their capacity of response to the will, but sooner than their nutrition. The conditions that are observed in these cases are the same as in the isolated form of neuritis, in which, although the primary change is in the connective tissue, the symptoms depend on the secondary changes in the nerve-fibres. Hence the statements made on p. 71 are equally applicable to multiple neuritis. The reactions often furnish important information, especially soon after the onset; the more the irritability deviates from the normal the greater will be the subsequent wasting, and the longer the duration of the palsy. When some faradic irritability returns in both nerve and muscle it may be regarded as a sure indication of advancing restoration of structure, which is certain to involve a progressive increase of conduction of voluntary impulse, and response to it. If the trunk nerves are not involved, the prognosis in severe cases is a little better when the arms are chiefly affected than when the legs have suffered most.

The sensory disturbance may be expected to follow a course on the whole similar to that of the motor symptoms. A diminution in the spontaneous pain may be anticipated sooner than in the tenderness of the nerves and muscles, which usually lasts, in lessening degree, until the muscles have regained some power. The extreme tenderness, indeed, passes away with the spontaneous pain, but some degree of over-sensitiveness continues for a long time. Happily the tendency to persistent neuralgia, seen after the isolated form, need not be anticipated after multiple neuritis. We find the explanation of this in the absence of the new formation of connective tissue from the inflammatory products, which in the isolated form perpetuate, by cicatricial contraction, the irritation to which inflammation gave rise.

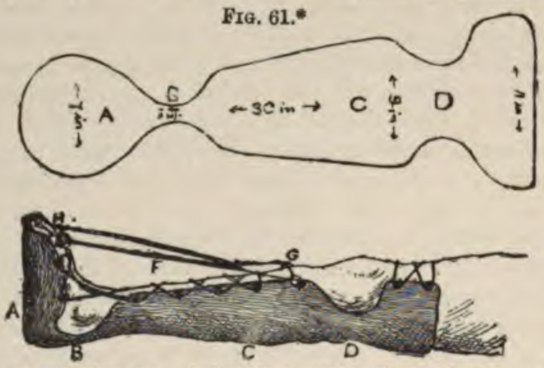
There is little difference in the prognosis, as regards recovery, in the different forms of neuritis. The anticipations that may be formed are governed in all by the rules just mentioned, and the risk to life has been already considered. In all, however, it may be repeated the extent to which the cause is under control forms an essential element in the problem, and must influence the conclusion in every case. The prognosis, however, is better in the sensory than in the motor form, better when the arms escape than when all the limbs are involved, and better in cases of chronic than of acute onset, and better if an apparently acute onset is really such than if it succeeds slight symptoms of longer duration. When the malady follows an acute specific disease, the severity of the latter is no guide to the course of its sequel; severe neuritis may follow a mild attack of the acute specific.

TREATMENT.—The different classes of multiple neuritis here considered need treatment that in some respects varies, and in others is the same in all forms. In all it is essential to discover, if possible, the cause of the neuritis, and to remove it or arrest the continuance of its action. This obviously should be the first consideration in cases due to alcohol, in which it is sometimes of great difficulty. It is desirable, as a rule, that alcohol in all forms should be given up entirely, but often this has to be done by degrees, a great reduction in amount preceding entire abstinence from it. With patients who are "their own masters" the reduction is often most difficult to secure, and it is necessary to give a warning that not only will life be in danger, but that the pains will certainly increase, and continued indulgence will entail sufferings so great that any sacrifice would be made, or deprivation endured, if their intensity could be realised. With every female drinker, deception should be considered not only possible but probable; those who are under the control of relations or of a husband have generally had a long training in duplicity in order to procure stimulants, and it is easy for them to continue it, even when kept in bed, if they are attended by their own servants. These are corrupted, and alcohol is taken in secret when the fact is being stoutly denied. If a case does not present the arrest or improvement that might reasonably be expected from the withdrawal of the cause, such continued drinking should always be suspected. Hence it is of great importance that such patients should be, whenever practicable, under the immediate care of trustworthy trained nurses, rather than of their own servants or of relatives who may be improperly influenced or persuaded; if the patient's means do not permit this, removal to a hospital or some public institution is desirable. When the heart is too weak to permit the entire withdrawal of stimulants, the amount taken must be regulated solely by the state of the pulse. In such cases total abstinence is attended with some danger of the cardiac failure which has been mentioned as one of the causes of death. When the disease is due to cold, the patient should be carefully preserved from risk of further exposure, which during the early stages may convert a slight into a severe attack. Even in alcoholic cases a chill will cause a rapid increase in the symptoms, and both this and fatigue should be carefully guarded against. In other forms, also, these measures, adopted in the stage when pains and tenderness are the only or chief symptoms, would often prevent the further development of the disease.

Physical rest is of great importance, and in all cases, except the slightest, rest in bed is desirable The excitation of the nerves caused by movement tends to intensify the morbid process in them, and whatever produces pain has the same effect. If the patient is in bed, local treatment can be more readily employed, and the restraint facilitates the necessary control over habits. The feeding of the patient is an important part of treatment, especially when the stomach has been deranged by alcohol. At the outset, warm fomentations should be applied over any tender nerves. Care must be taken in their use over the region supplied by the affected nerves, on account of the danger of vesication; troublesome ulcers may be caused by even moderate degrees of heat, as in the case mentioned on p. 79. Such applications, and also counter-irritants, have less effect in the parenchymatous forms than when there is a tendency for the connective tissue and sheath to suffer, and for their vessels to be involved in the process. A warm bath, for fifteen or twenty minutes daily, is often of service if the patient is able to bear the necessary procedures.

Care should be taken that the patient does not habitually assume a posture that will promote the occurrence of deformities The dropping of the feet is the greatest danger, and should be prevented by a large and heavy sand-bag about nine inches in diameter, which may be adapted to the position of the feet, or by a board placed across the bed. Frequently such a boot as that figured (Fig. 61) is of great service in preventing or minimising contraction of the gastrocnemii. Another danger is the habitual flexion of the knees, permitting a contraction of the hamstring muscles, and due to the discomfort that is caused by extension of the knees; this should, however, be insisted on, because the initial discomfort soon passes away if extension is adopted from the outset. The extension of the knees involves that of the hip-joint, and prevents contraction of its flexors.

FIG. 61.*



No drug has any very marked influence on the morbid process. It has been seen that multiple neuritis tends to lessen after its cause has ceased to act, and under such conditions it is easy to come to the conclusion that drugs do good which merely do not hinder. Mercury, which is certainly useful in isolated perineuritis, has little influence on the parenchymatous form, but it may be tried in any case in which it seems probable that the sheaths are primarily affected, and in which pain or tenderness in the larger nerve-trunks is a prominent symptom. During an acute febrile onset the treatment should be that adapted to the general state, and must differ according to its cause. In such an onset of an alcoholic neuritis, citrate of potash, nitrous ether, and compound tincture of cinchona are suitable, with a little digitalis if there is feebleness of the pulse. When due to cold, salicylate of soda may be given, or, if the attack has been predisposed to by alcohol, salicylate of potash may be substituted so as to avoid the retention of uric acid in the system by the soda. The probability of a gouty state of the system should be remembered in all cases in which there is a history of alcoholism. In the toxæmic forms in which there is a suspicion of a blood-state allied to that of septicæmia, no drug affords such a prospect of influence as the tincture of the perchloride of iron in full doses, 20—30 minims three or four times a day. Afterwards, when the actual onset is over, and from the first in the more chronic cases, tonics answer best—iron, quinine, or small doses of strychnine. Iodide of potassium, for some reason, seems to do good only in the chronic sensory form. In this also arsenic has been found useful, but arsenic should be given with caution, and in small doses; there is some danger that large doses may intensify the malady, since polyneuritis is one of the effects of chronic poisoning by arsenic, and has actually been produced by the medicinal use of the metal. Cod-liver oil is useful in the later stages, especially if there is impairment of general nutrition. In alcoholic cases the addition of cocaine, $\frac{1}{12}$ to $\frac{1}{4}$ grain to each dose of whatever is taken, aids in lessening the craving for stimulant. When due to malarial poisoning, quinine may be given freely. The complications of multiple neuritis do not need other treatment than that suitable to them when they occur alone, nor do they indicate any special modification in the treatment of the disease of the nerves.

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* For full description of this simple apparatus, see 'Clinical Journal,' March 6th, 1895.

In most cases the pain is such as to render anodynes necessary. Of these, morphia, by hypodermic injection, is the most effective, but should be employed only as a last resource, and so regarded will not often be found necessary. Its danger is the readiness with which those persons who were previously alcoholic become habituated to it. It may be given by the mouth with less risk, but in other drugs we may find a substitute that is effective in most cases. Injections of cocaine beneath the skin over the seat of pain often afford considerable relief, and cocaine may thus be given instead of by the mouth, securing the double object of lessening at once the local pain and the craving for stimulant—which is relieved almost as effectually when cocaine is given by the skin as by the mouth. Atropine may also be given hypodermically. In antipyrin and acetanilide we have drugs of another nature, that often give great relief to spontaneous pain. Indian hemp may also be given regularly without harm, and without interfering with the occasional administration of other anodynes; while to procure sleep, if pain is slight, reliance may be placed on bromide, sulphonal, trional, or chloralamide. Antipyrin also often gives a patient good night in spite of some pain. When there is mental excitement hyoscine is useful, especially by hypodermic injection in doses of $\frac{1}{400}$ to $\frac{1}{150}$ or $\frac{1}{100}$ of a grain.

The application of electricity to the paralysed muscles is important in order to maintain their nutrition in as good a state as possible during the process of recovery of the nerves. Only voltaism has the power of doing this, and should be applied daily, large sponge-holders being employed so that the current may reach as much muscular tissue as possible; only such a strength should be employed as will produce visible contraction and cause no after-pain. Voluntary muscular action constitutes a more effective stimulus to nutrition; but if, although the muscle can be put in action by the will, there is loss or great diminution of faradic irritability, or an excess of voltaic irritability shows that some fibres are in an abnormal state in consequence of the degeneration of their nerves, voltaism may still be applied with advantage. There is no evidence that the application of electricity to the nerves has any influence on their regeneration. Slight sensory loss is sometimes lessened in the chronic stage by the application of faradism by the wire brush.

The tender limbs may be wrapped in cotton wool, with or without a covering of oiled silk. Massage is of service in the later stages of the disorder for its influence on the nutrition and circulation in the affected limbs. An upward movement of the pressing hand helps the circulation of fluid in the vessels and in the tissues. It cannot be borne during the acutely painful stage, and, indeed, as long as it gives pain it probably has more capacity for harm than for good. In the later stages it helps to overcome the contracture of the muscles, which should be gently extended at the same time as they are rubbed, —pressure, for instance, being made upon the ball of the foot at the same time as the calf muscles are rubbed.

The very long course of all severe cases makes a heavy demand on the patience of the sufferer and the perseverance of the medical practitioner; but the prolonged convalescence has the advantage, in alcoholic cases, of enabling a habit of abstinence to be formed. This, with the recollection of what has been endured, renders multiple neuritis more often a cure of intemperance than any other of the many maladies to which alcohol gives rise. During the slow recovery, the measures above indicated—tonics, electricity, and massage—should be continued. As already stated, the power of standing is interfered with for a time after the muscles have regained adequate strength, by the contraction of the gastrocnemii, causing a degree of talipes equinus. This is often so considerable as to suggest the desirability of dividing the tendo Achillis, but the operation is seldom if ever necessary. The attempt to stand and walk constitutes a powerful means of extension of the calf muscles, before which they soon yield sufficiently to permit the balance of the body to be maintained, and then progress becomes more rapid. The contraction at the knee and hip is more difficult to get rid of, but generally yields in time to persevering and gentle efforts.

ACUTE MULTIPLE MYOSITIS. ACUTE POLYMYOSITIS.

IN rare cases there occurs an acute interstitial inflammation of the muscles, which presents a general resemblance in distribution, and in toxic causation, to the acute polyneuritis we have just considered, but differs in the facts that the affection of the nerve-fibres is trifling and evidently subordinate, and that the subcutaneous fibrous tissue is usually also inflamed, causing œdema and erysipelatoid dermatitis. This is sometimes absent while the nerves are occasionally inflamed in greater degree, precluding an absolute separation, although most cases present a sufficiently uniform difference to make a distinctive type. The malady is so uncommon, and the cases that have been met with have varied so much in severity and precise causation, that they hardly do more than illustrate the variety of special form which may be assumed by such toxic maladies, and which indicate corresponding features in the poison which causes them. The symmetry of its manifestations is conclusive evidence that the blood is its vehicle, and the facts that a source of definite poisoning can often be traced, while the distal parts of the limbs are commonly the first to suffer, constitute a conspicuous connection with polyneuritis.

Although not a primary affection of the nervous system, its relations justify its mention here, especially since its features have enough in common with the acute form of multiple neuritis to involve some risk of confusion in diagnosis.

ENDEMIC NEURITIS.

MALARIAL NEURITIS.

The clear evidence that has accumulated, showing the dependence of multiple neuritis on toxic blood-states, some connected with an organismal virus, prepares us to find that it often results from the products of the growth of organisms of endemic character, such as cause the malarial fevers of which we are able to discern a variety of forms. Conspicuous among them is the malady known generally by the name of "beri-beri," but a simpler form of neuritis palsy is more common, although absolutely infrequent. Various forms, however, result from the untypical varieties of malarial disorder, and their occurrence, causation, and relations are still only imperfectly discerned, met with, as they are, chiefly in tropical countries.

The characteristic form involves chiefly the motor nerves, usually of the legs, and is met with especially in India and on the coasts of Africa. The arms suffer only in severe and prolonged cases. It is usually subacute in course, and the resulting disability entails early removal from the causal conditions, which lessens much the danger to life that is involved. Secondary contracture in the muscles is especially common, and constitutes a troublesome sequel, prolonging for months the inability to walk, unless prevented by the means described elsewhere.

Sensory symptoms of various characters may be the leading features in other cases in which the organisms are peculiar in their nature, and these give rise to symptoms which are prone to set up special difficulties in diagnosis, unless their probable nature is suspected, and the significance of the exposure to the cause is perceived, and receives due weight.

BERI-BERI.

Beri-beri, the Kak-ké of Japan, is also known by various popular names in the several countries of which it is native.[*] It is a widespread disease, passing by a contagion that is readily conveyed and propagated, having apparently its chief homes in Japan, the Eastern Archipelago, India, New Zealand, Ceylon, the South Pacific Islands, and the coast of Brazil. It is especially prevalent in the Dutch East Indies, among the soldiers and in the prisons, and this led to its systematic investigation under the direction of the Netherlands Government. Through this our knowledge of the malady was much increased, especially by the investigations of Pekelharing and Winkler,[†] who had a large number of cases under their observation both during life and after death. Still more recently a troublesome outbreak occurred at a lunatic asylum at Dublin. Opinion as to its nature and cause has varied much, and still is far from uniform among those who have studied it; but there is a strong preponderance of evidence that it depends on a specific organism, and that symmetrical peripheral neuritis is the common effect of the virus, and the mechanism by which its chief symptoms are produced. The organisms found[‡] are in the form of rods and cocci, but it is probable that these are only different stages in the development of the same species. They have been cultivated, and peripheral neuritis of nearly the same distribution as in beri-beri has been produced by their inoculation. Repeated inoculations are, however, necessary for this result to be produced; and hence, from this and the phenomena of the disease, it is assumed to be not a simple infectious malady, capable of being induced by a single exposure to its cause, but one in which repeated opportunities for infection are necessary. In harmony with this are the facts that sufferers acquire the disease from residence in certain infected houses. or places in which it is supposed that the soil is saturated with the organisms; that they may rapidly recover on removal to a district that is free, and relapse only on returning to an infected place; that it prevails where persons are gathered together, as in barracks and prisons; and that the air of these places is found to contain the organisms which, collected from it, will cause the disease in animals, from whom it is transmissible to others by further inoculation. The serious extent to which it may spread among those who live together under conditions favouring its extension, is shown by the instance of a ship arriving in Japan from New Zealand after a voyage of 272 days. The disease spread on board so rapidly that altogether 169 cases occurred, with twenty-five deaths. In 1878 no less than 38 per cent. of Japanese soldiers were affected. It is probable that the inhalation of dried organisms floating in the air as part of "dust," is the chief way in which the disease is propagated. By some it has been thought that a nitrogenous diet induces or predisposes to the disease, and facts apparently supporting the opinion have been adduced; an exclusive fish diet has been thought to cause it, and so also has a rice diet. A remarkable outbreak in Manila in 1880 followed a period of rice-eating during a cholera epidemic. These influences probably merely produce susceptibility.*

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[*] Beri-beri is probably a modification of the Cingalese name for the disease, bahrbari = extreme weakness. Kak-ké is the old Chinese name for it, by which it is mentioned in their medical works since 200 B.C.; it is derived from two words, meaning "legs" and "disease." The affection disappeared from China two centuries ago, remaining in Japan, where it continues a most serious endemic malady.

[†] Pekelharing and Winkler, 'Onderzoek naar den aarden de oorzaak der Beri-beri,' Utrecht (sep. publication), 1889, analysed in the 'Centralbl. f. Nervenkr.,' 1889; also 'Weekblad f. Nederl. Geneesk.,' 1888, and 'Deut. med. Wochenschr.,' 1888, No. 30. Other important contributions to the subject are by Bälz, 'Zeitschr. f. kl. Med.,' 1881; Scheube, ib., 1882; Weintraub, 'Wien. med. Wochenschr.,' 1888, Nos. 23—44; Van Eecke, 'Tijdschr. v. Nederl. Indre,' 1887, p. 71; Wernich, 'Virchow's Archiv,' Bd. lxxi; Miura, 'Virchow's Archiv,' Bd. cxi and cxiv, whose conclusions, however, differ from those of most investigators; Seguin, 'Phil. Med. and Surg. Rep.,' 1888; and Springthorpe, 'Australian Med. Journal,' 1889. Bälz, in 1881, expressed the opinion that the disease was a specific "panneuritis."

[‡] First described by Bälz and Scheube, and since by Pekelharing and Winkler, Eigkman, Weintraub, Springthorpe, and others.

It is a remarkable fact that Europeans seldom suffer. Males are more liable to the disease than females, and it is chiefly prevalent during the hot season.

Symptoms.—Peripheral symmetrical neuritis is a constant feature of the malady, which is usually chronic in course and gradual in onset, but prone to undergo acute exacerbations. These have been generally regarded as acute forms of the disease, but it is said by Pekelharing that the symptoms of neuritis may always be discovered before the onset of the definite symptoms, and this when the patient is ignorant of their existence and feels quite well. Most observers have failed to recognise this, and it is probably true only under certain conditions, and persons suddenly exposed to an intense infection may suffer acutely from the very commencement. Occasionally the malady develops with extreme rapidity and severity. On the other hand, many cases are chronic throughout, and last for months. The leading symptoms are those of multiple neuritis (chiefly affecting the legs and the cardiac branches of the vagus), dropsy, and symptoms of cardiac failure. The amount of urine is generally lessened, and its secretion may be almost suppressed in acute cases. A "critical" increase in the secretion may mark the commencement of improvement. It is unchanged in character, and does not contain albumen. The dropsy is a very variable symptom, and this has led to the distinction of two forms, the dry and the dropsical. Pekelharing and Winkler, however, found some effusion of fluid almost invariably after death, and it is probable that its conspicuous presence or absence depends chiefly on the state of the heart (conditioned by that of its nerves), and partly on the trophic and vaso-motor disturbances due to the local neuritis. One very constant symptom is said to be a disagreeable epigastric sensation, variously described as pain, dragging, &c., sometimes very severe.*

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* Putnam ('Boston Med. and Surg. Journal,' 1890) and Birge, in the same periodical, describe a form of neuritis very like beri-beri occurring in men long at sea engaged in fishing, with poor food. In some of the cases scurvy was also present.

The earliest symptoms are a change in the electrical excitability of the peroneal nerves and the flexors of the ankles (which suffer most throughout)—a slight degree of the reaction of degeneration, quantitative and often qualitative. These are often to be found before there are any subjective symptoms, which begin as a sense of heaviness of the legs, readiness of fatigue, dysæsthesiæ and diminution of tactile sensibility in the lower limbs, palpitation and undue excitability of the heart. The electrical changes may be met with in slight cases which proceed no further,† and have shown that some patients, supposed to be shamming, were real sufferers. To these subjective symptoms are added other objective signs,—œdema along the edge of the tibiæ, a peculiar pasty and stiff aspect in the face, an increase in the cardiac dulness to the right, roughness of the first and accentuation of the pulmonary second sound. These symptoms may increase slowly, or rapidly in the form of an acute stage. The degenerative reaction becomes complete in the muscles first affected, and they become paralysed and undergo the characteristic atrophy, while other nerves and muscles progressively suffer in the same manner,—the calf muscles, the extensors of the knee, the adductors of the thigh, and, lastly, the flexors of the thigh and the abductors of the hip. In the trunk the abdominal muscles and intercostals may be involved, and in severe cases the arms are paralysed, first the extensors of the wrist and fingers, later the flexors, and sometimes most of the muscles, may be so atrophied as almost to disappear. The face often suffers: the diaphragm may become paralysed, and also the larynx (the inferior before the superior laryngeal nerve); while grave cardiac weakness and increasing dilatation of the heart testify to the serious implication of the cardiac branches of the vagus. Simultaneously, sensory symptoms develop, corresponding in distribution to the more severe motor symptoms; sensibility to touch is lessened or lost (first on the inner side of the lower limb), while that to pain usually remains, and sometimes is augmented to the degree that constitutes what has been termed "anæsthesia dolorosa." The temperature-sense may be diminished to either heat or cold or both in various parts, and there is often considerable loss of cutaneous sensitiveness to faradism. There may be tingling, formication, and other dysæsthesiæ, together with tenderness of the nerves and muscles, but far less in degree than in most forms of polyneuritis, and chiefly marked in the early stage of the disease.

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* Von Tuzelmann, 'Lancet,' 1894, vol. ii, p. 1467.

† Pekelharing and Winkler, Eigkman (1889), &c.

The œdema that is so common begins in the legs, usually spreads widely, and involves not only the subcutaneous tissue but the cavities of the peritoneum, pleura, and pericardium: in the last it seriously impedes the action of the already dilated and feeble heart. It is to cardiac failure that death is commonly due; the other chief cause is failure of the respiratory muscles, aided generally by effusion into the pleural cavities, and accompanied by the indications of cardiac weakness, and of dilatation of the right side of the heart. From this cause the cardiac dulness sometimes increases rapidly, even in a few hours. The œdema is usually attended by increasing anæmia, which doubtless facilitates its occurrence. These symptoms are always later in development than are those due directly to the neuritis. In the most acute cases the definite symptoms take commonly six or eight weeks to run their course, and the usual duration of the malady is several months. Death from the heart failure is usually prolonged, and accompanied by much distress. Occasionally a very acute form prevails, in which rapid failure of the heart and various effusions lead to death in about a fortnight, and cause a mortality of 60 and 70 per cent.

Pathological Anatomy.—The only constant changes are in the nerves, but some serous effusion is almost invariable. Thus Pekelharing and Winkler found an excess of fluid in the cellular tissue in 83 out of 85 cases—about 97 per cent. The pericardial fluid was increased in 67 per cent. of the cases, in about 23 per cent. there was hydrothorax, and ascites in 14 per cent.

The changes in the nerves are often indistinct to the naked eye, although always conspicuous under the microscope. They are met with in both motor and sensory branches, and, in advanced cases, in the nerves of the vessels. They are always greatest at the terminal portions, lessening upwards, while the nerve-roots and spinal cord are always normal, with the doubtful exception of vacuolation of the motor cells,* and some degeneration of the fibres of the posterior column in a few cases.† The first change is an increase in the segmental nuclei, shrinkage of the medullary sheath at the nodes, and a

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* Miura, loc. cit.

† Van Eecke, loc. cit.

peculiar "lumpy" degeneration of the medulla, sometimes replaced by a "foam-like" appearance in it, concealing the axis-cylinder. Both changes are often accompanied by the formation of granule corpuscles. These slighter changes are found chiefly where the lesion is commencing, and especially in the vagus and phrenic nerves, in which, necessarily, a considerable degree of change is incompatible with life. They have been seen in the branches within the heart, in some of the fibres of the trunk, and in the laryngeal nerves. Where the change in the nerves is more advanced, the white substance is entirely broken up, and the axis-cylinder presents segmentation or irregular swellings, or has disappeared, and the increase in the nuclei of the neurilemma sheath is still greater than before. The blood-vessels of the nerves are but little changed in the early stage; after a time they may present some thickening of the wall, accompanied by a general increase in the amount of the interstitial tissue; but, as a rule, there is an absence of any signs of interstitial inflammation—the changes are essentially parenchymatous.

In the muscles there may be at first a cloudy opacity with indistinctness of the transverse striæ, going on to granular degeneration; or the fibres may simply become narrow, preserving their striation. The nuclei of the fibre-sheath are always increased in number, but the interstitial tissue is generally but little altered.

Pathology.—The characteristic organisms have been invariably found in the blood, and, what is a remarkable instance of their slow action, and of the cumulative influence needed to generate the malady, Pekelharing and Winkler* found them in the blood of all the inhabitants of an intensely infected place, whether these had or had not symptoms of the disease, and failed to find them in the blood of persons living in places free from infection. Cultures from the blood reproduce the malady, and so do cultures from the animals thus infected. The organisms have not been found in special association with the morbid process in the nerves. It thus appears that the malady bears a close resemblance, in its essential features, to cases of multiple neuritis due to a chronic blood-poison such as alcohol, and still more to that which, as we shall see, is presented by chronic arsenical poisoning. There is a slow accumulation in the blood of a virus that has a specific action on the peripheral nerves, and like some others, first and chiefly on the nerves of the legs, but with a greater affinity for the vagus, especially for its cardiac branches, than most others. Although invariably associated with the presence in the blood of a special organism, the absence of this in the vicinity of the lesion, taken in conjunction with modern researches in bacteriology, makes it probable that the virus is a chemical substance produced by the organism in its growth. This brings the disease into close affinity with some other forms of multiple neuritis, on which it throws sufficient light to justify this somewhat full account of a malady that is not likely practically to concern many readers. If we regard, as apparently we must, the cardiac failure as the result of the cardiac neuritis, and the dropsical effusion as the combined result of this and the local trophic and vaso-motor influence of the neuritis (to which, perhaps, the change in the blood should be added), we have a remarkable illustration of the extent, variety, and degree of the secondary processes of disease which multiple neuritis may produce.

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* Also Ogata (1888) and others. They have also been found sparingly in the organs, even in the spinal cord, which is commonly free from disease.

The *Diagnosis* can only be a matter of difficulty in a very early case, or in a patient who is not known to have been exposed to the infection, or in consequence of ignorance of the disease. Doubt may exist, however, whether in a given case the malady is alcoholic neuritis or beri-beri. The former is probable if the arms are involved at the same time as the legs, still more if they suffer first; also if pains are severe, œdema absent, and the heart unaffected. The converse would make the endemic malady probable. Beri-beri has certainly been intensified by alcohol in some cases, and the symptoms then presented are a combination of the two,—pains, associated with the weakness and enlargement of the heart and the widespread tendency to dropsical effusion met with in beri-beri. In most cases some other indication of the influence of alcohol, especially enlargement of the liver, will assist the diagnosis. From other forms of multiple neuritis it is unlikely that the diagnosis would have to be made. From affections of the spinal cord the distinction (chiefly needed in the chronic "dry" variety) depends on the points already mentioned in the account of multiple neuritis. Acute cases with much œdema and weakness of the heart, and enlargement of the right side, may be mistaken for primary heart disease; but if the symptoms of beri-beri are known, the affection may be recognised by the absence of any indications of primary valvular disease; while an examination of the legs will reveal the weakness of the flexors of the foot and lessened tactile sensibility in the skin over them.

Treatment.—Beri-beri is one of those diseases for which it is probable that a method of at least prophylactic inoculation will be discovered in the future. At present the destruction of the organisms by disinfecting agents, or, better, the removal of a person to an uninfected district, constitutes the only means of preserving him from the continued entrance of the poison—a measure essential for successful treatment, and, in many cases, all that is really necessary. The limits of disinfection are, however, reached with the dwelling-house; and the great difficulty presented by the disease is the manner in which whole districts are capable of giving rise to it, probably by the infection of the soil, which cannot be rendered innocuous. However, even within the narrow limits of personal use, disinfection has achieved some remarkable results, and in few diseases has the light of pathology given more useful guidance to the hand of the practitioner

In most respects the treatment of the symptoms of beri-beri does not differ from that of alcoholic neuritis. A special point, however, is that the strengthening of the heart by digitalis or strophanthus is more often of urgent importance, and requires greater care lest the feeble fibres be overtaxed. Small doses should therefore be given—three or at most five minims of the tincture of digitalis. Bodily rest is of paramount importance to the weak organ, in order that there may not be any demand on its energies for other work than that involved in the maintenance of what may be termed static nutrition, and that it may be spared the increased work necessitated by muscular exertion. Strychnia, quinine, &c., may do good, but their use in this disease does not differ from that in the more common forms of multiple neuritis.

Leprous Neuritis.

Far removed as leprosy may seem to be from the malady last described, both by its general character and extremely chronic course, it is certainly also an endemic disease; its cause must be sought in local influences; and it agrees with beri-beri in the nature of those influences. Since the researches of Carter, Hansen, and Neisser showed the constant presence of a specific bacillus in all the new formations met with in the disease, their observations have received abundant corroboration; the disease was indeed one of the first to be proved to depend on living organisms. The virus resembles that of beri-beri in being transmissible from individual to individual, but differs in its capability of passage by inheritance, in the less readiness with which it passes from person to person, and in the extreme slowness with which it commonly develops to the degree necessary to cause symptoms. Years may elapse after the last possible exposure, before distinct symptoms of leprosy appear. In the case from which Fig. 63 was taken the sufferer was a European of healthy parentage, who could not have been infected within ten years of the first symptoms. In one case of infection from adult to adult, nine years elapsed after the death of the leper before the man who had lived with him began to suffer. When, indeed, a large quantity of the virus is received, the malady develops much more rapidly. But leprosy differs from beri-beri, and from most other forms of multiple neuritis, in the fact that although inflammation of many nerves is frequently a part of the disease, it is not an essential part or a constant feature of the affection. On the contrary, it is only one of an extensive series of lesions, inconstant, not only in its occurrence, but in its seat and extent. It characterises a special variety of the disease, the "anæsthetic leprosy," in which not only areas of the skin become insensitive, but the ends of the fingers may suffer such an impairment of their nutrition that they are lost. Moreover, as we have seen in the introductory section, the want of strict symmetry is associated with an important difference from the symmetrical forms—the neuritis is not a parenchymatous or degenerative form, but is a perineuritis and interstitial neuritis, and the damage to the fibres is secondary. This change in the connective tissue is associated with the actual presence of the organisms in it. The neuritis thus seems to be due to the direct action of the bacilli on the affected tissues, and not, as in beri-beri, to a virus circulating in the blood—not to a product of the organisms, but to the organisms themselves. This fact enables us to understand better its peculiar features, and especially its irregular distribution; the inflammation occurs where the organisms happen to fix themselves in the connective tissue, and although certain common conditions on the two sides may determine a partial correspondence in the nerves affected, there is not the symmetry that results from an action on like structures on the two sides, equally accessible to the virus. In this, and in its connective-tissue seat, the neuritis of leprosy resembles more the isolated neuritis of syphilis, rather than the common varieties of the multiple form.*

The symptoms of the malady are extensive and various, but only those that consist in derangement of the functions of the nervous system concern us here, and it is alike needless and impracticable to describe the others. The nervous symptoms of leprosy depend almost exclusively on inflammation of the nerves. These symptoms are muscular wasting and anæsthesia, greatest in degree towards the extremities of the limbs, but more or less irregular in situation, and in the region of the distribution of nerve-trunks, which, if accessible, are obviously diseased. The symptoms are not confined to the limbs, but are also met with in the face, where an affection of the fifth nerve may cause anæsthesia, and movement may suffer from involvement of the trunk of the facial nerve, so that the eyelids cannot be completely closed. The form of sensibility that is impaired also varies; sometimes touch, sometimes pain is lessened in greater degree, but usually all forms are implicated to some extent. The legs often suffer before the arms, but a special order of affection cannot be distinguished. There may be unpleasant tingling sensations, but pains are usually trifling, perhaps because the process is extremely chronic. In some cases there may be severe pain in the position of the nerves or deeply seated in the limbs. The inequality of the affection of the fibres of the same nerve is shown by the fact that the knee-jerk may persist although the extensors of the knee are partially paralysed. Fig. 62 shows the distribution of anæsthesia in the hands in a well-marked example of this disease which came under my observation some years ago. The patient was a creole of Mauritius, who had come to England in early childhood, and presented the first distinct symptoms at twelve years of age—changes in the pigmentation of the skin, anæsthesia, and muscular wasting. They steadily increased during the next four years, the anæsthesia extending each half-year from one finger to another. There was also patchy anæsthesia of the legs below the knee. As the figure shows, the loss of sensation does not correspond in area to nerve distribution. This depends on the fact that even when all the nerves, including those of the fingers, are diseased, all the fibres may not be destroyed in any one nerve, so that some sensibility exists in each nerve area. Various perversions of sensibility, spontaneous "dysæsthesiæ," tingling, formication, and the like, may precede the loss. The latter usually occurs gradually, but sometimes so suddenly as to suggest some secondary vascular lesion, hæmorrhage or thrombosis, in the vessels of the nerve that is the seat of the process—a possible result in its early and most active stage. It must be remembered that in all inflammations of the sheath and interstitial tissues this proceeds to a considerable degree before the fibres of the nerve suffer, and thus sudden loss of sensibility does not show that the nerve was previously healthy. This occasionally sudden onset is of much practical importance; one case was sent to me from a place in which the disease is endemic, by a doctor well acquainted with it, who had not suspected leprosy because the anæsthesia came on almost suddenly.

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* Gerlach ('Virch. Arch.,' cxxv, 'Neur. Cent.,' 1891, p. 464) says infection is from skin, branches of nerves being then affected and finally the nerve-trunks, so that skin may be seen infiltrated when nerves still remain free.

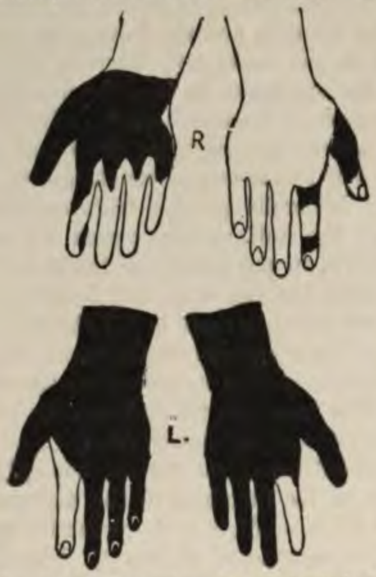

FIG. 62.—Distribution of anæsthesia in a case of leprosy. The black areas indicate the loss.

Accompanying anæsthesia there is usually some muscular wasting, which may be great. In the case figured it was considerable, although only in the small muscles of the hands was it comparable to that of progressive muscular atrophy. The electrical excitability of the muscles was greatly lowered to each current, and this is perhaps the most common condition; either the damage to the nerves is so chronic that the fibres undergo slow changes *pari passu* with those in the nerves and their endings, or the patient comes under observation at a late stage in the local affection, at which the changes present in the early stage have disappeared; sometimes, indeed, all irritability has vanished. In cases that are seen soon after the onset of the lesion of the nerves, there may be the reaction of degeneration in the muscles, either partial or complete, and there is often a conspicuous exaltation of the mechanical irritability of the nerve-trunks. When the facial nerves are affected this may be well seen, and may resemble that met with in tetany.* The thickening of the nerve may be felt when that which is affected is accessible. The state of myotatic irritability varies according to the distribution of the neuritis; it is lost at places where the sensory or motor fibres are involved, but those supplying the front of the thigh and its muscles often escape, and the knee-jerk is preserved. Anchylosis of the joints may occur, as in other forms of neuritis.

The "mutilations" of leprosy, by which the ends of the fingers and toes are lost, are also regarded by some as a consequence of the neuritis. The numerous other symptoms of the disease are independent of the nerves (except perhaps the pigmentation of the skin), and are beyond the province of this book.

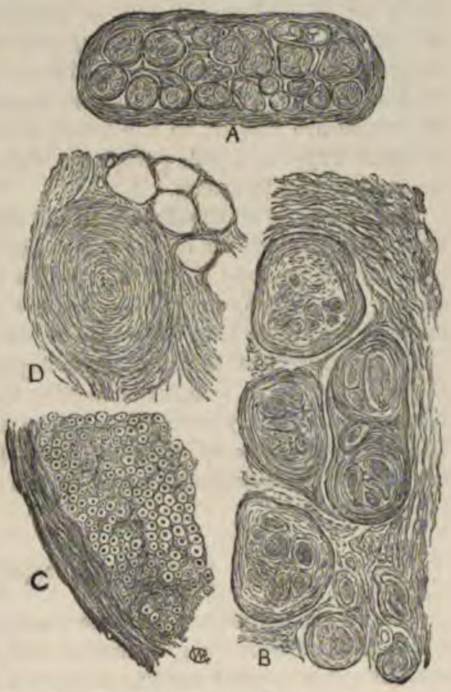

FIG. 63.—Sections of nerves from a case of anæsthetic leprosy, under the care of Dr. Buzzard. A, median nerve at wrist × 5; B, portion of same more highly magnified; C, part of a less diseased fasciculus from the ulnar nerve; D, a small fasciculus from median in which the concentric fibres have invaded the whole area of the fasciculus.

The neuritis of leprosy is typically adventitial (Fig. 63). The primary sheath and the secondary sheaths of the fasciculi are greatly increased in thickness, and consist of nucleated fibrous tissue arranged concentrically (A, B). From the sheath, tracts extend into the interior of each fasciculus (B), isolating the groups of nerve-fibres. The increase of tissue even extends between the fibres themselves (C), and these undergo slow wasting; many of the fibres in the figure are seen to be distinctly narrower than normal. The concentric growth of fibrous tissue may even invade the whole area of the fasciculus, all the nerve-fibres perishing before it (D). The characteristic bacillus of leprosy is found abundantly, in recent cases, in the new tissue of the nerves. Peculiar cells are met with, infiltrated with the organisms. As the fibrous tissue develops and contracts, the bacilli seem to perish, and ultimately can no longer be discovered.

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* Schultze, 'Deut. Arch. f. klin. Med.,' 1888, Bd. xliii.

The diagnosis depends on the occurrence of irregular areas of anæsthesia, generally associated with irregular patches of pigmentation and pallor in the skin, and often with muscular atrophy, in a person who has been exposed to the risk of infection, generally by having lived in a district in which the disease is endemic. The irregularity of distribution and the limitation of considerable sensory changes sufficiently distinguish it from other forms of neuritis. The spinal affections with which the mixed anæsthesia and wasting are most likely to be confounded are, as Schultze has pointed out, cases of syringomyelia in which the distension of the central cavity damages the grey matter, and may cause similar symptoms of irregular distribution—muscular atrophy, sensory impairment, trophic disturbance.* But these are limited to the arms; and if the legs suffer it is in a different way—as simple paraplegia with excess of myotatic irritability that may go on to spasm.

The prognosis of the disease in the simple anæsthetic form is grave only when the sufferer is still exposed to fresh infection, or in cases of considerable severity. But it must be remembered that, just as the disease may develop long after exposure to its cause, so it may slowly increase for a long time after this exposure has ceased.

The treatment of the affection is beyond the scope of this work; in so far as the nerve symptoms require special measures, these are the same as in ordinary neuritis. A trial may be made of the stimulation of the muscles by whatever form of electricity they will respond to, and of the anæsthetic areas in the skin by the wire brush and faradism.

HERPETIC NEURITIS.

Among the cutaneous eruptions occurring in acute neuritis, that which characterises "herpes zoster" has been mentioned. This affection always presents a correspondence in area with nerve distribution; a definite neuritis has been found in the rare cases which have afforded an opportunity for pathological investigation; and pain with other nerve symptoms is common, and may be conspicuous. Hence herpes, although usually described as a skin disease, is really the manifestation of a form of neuritis which is sufficiently individual in course and features to merit a separate description.†

Causes.—Our knowledge as to the ætiology of the neuritis which underlies the most common form of herpes is still very incomplete. An important clue is, however, afforded by the fact, first pointed out by Mr. [illegible] Hutchinson, that the disease may be induced by the presence of arsenic in the blood. This indicates that it may be due to a chemical toxic agent in the circulation, and suggests that as its common cause. But the nature of the agent is still quite unknown. The careful researches of Rendu cast doubt upon the ætiological importance once attributed to gout, although a causal connection can occasionally be traced, as in a well-known case of Trousseau's. Ebstein * records the case of two brothers who, after eating mussels, developed the one urticaria, the other herpes. The affection has been traced to malaria by Winfield.† We know, furthermore, that some of the many varieties of rheumatic toxins may act upon various parts of the nervous system, in a manner not unlike that seen in poisoning by arsenic, so far as concerns the characters of the nerve inflammation. Herpetic neuritis may thus probably be produced by toxins introduced from without, or manufactured within the body; it may even assume an epidemic form. We do not know why it should usually be unilateral and show a predilection for certain regions; the reason is probably a local one, and it may be noted that herpes has been seen to develop in the scar of a burn, and to reappear in those left by a first attack forty-six years previously. Neither age nor sex seems to have much influence in relation to the disease.

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* See [illegible]'s Disease," later.

† [illegible] as to the nervous origin of herpes seems to have been made [illegible] of Medical Cases,' 1831, vol. ii, part 1.

Pathological Anatomy.—The indications of inflammation have been found especially in the ganglia on the posterior spinal roots; they tend to spread centrally towards the cord, rather than peripherally. The Gasserian ganglion, which corresponds to that of a posterior root, is similarly affected in herpes frontalis. Although these inflammatory changes were first described (by von Bärensprung) as long ago as in 1861, our knowledge as to their exact nature is still meagre. We can state, however, that they affect primarily the intercellular material, and that they are ordinarily very severe, frequently inducing hæmorrhagic exudation; the swelling and injection of the ganglia and adjacent nerve-trunks are usually macroscopically evident. We must distinguish from this primary form those in which the eruption is only a conspicuous symptom of inflammation communicated to the nerve, or clearly secondary to some other cause. Thus Wagner records in one case purulent pachymeningitis and swelling of the spinal ganglia of the affected area. The occurrence of bilateral herpes is explained by Kaposi ‡ on the ground of primary affection of the spinal cord, but no anatomical proof of this is adduced, and it seems more reasonable to look to the general action of the toxic agent in the blood as the cause. As to the nature of this agent we are, except in the case of the special poisons already mentioned, absolutely ignorant. This point has already been discussed in connection with neuritis.

With regard to distribution, although practically any sensory nerve

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* 'Virchow's Archiv,' vol. cxxxix.

† 'New York Med. Journ.,' 1895.

‡ Eulenberg's 'Real Encyklopädie,' art. "Herpes."

[illegible] is upon the intercostals, and particu[illegible] dorsal region; the fifth nerve, in any of [illegible] the first and second, and the branches of [illegible] to the occipito-cervical form, the small [illegible] nerves being most affected. The lower [illegible] seat; the arm very seldom suffers. [illegible] may spread to the adjacent motor nerves [illegible] spinal cord, leading in one case to death from [illegible] The herpes itself is commonly ascribed to im[illegible] but Ebstein (loc. cit.) regards it as due to [illegible] of the vaso-dilator fibres.

[illegible] of the herpetic eruption falls properly [illegible] dermatology. Areas of congestion appear [illegible] into inflammation with effusion; the epi[illegible] may ultimately form. The conspicuous [illegible] is probably to be referred to the [illegible] divisions, or their corresponding ganglionic [illegible] general toxic cause that the attack may be [illegible] prodromata, particularly vomiting, with con[illegible] neuritic symptoms the most conspicuous is [illegible] considered under the head of neuralgia; [illegible] distinctions between the pre- and post[illegible] which usually abates with the appearance [illegible] both more severe and of longer duration. [illegible] of the region served by the affected [illegible] eruption; there is in some cases per[illegible] conduction. Motor affections are also not [illegible] simply immobility from hyperæsthesia or [illegible] of the neuritis to motor nerves. In the [illegible] muscular atrophy with more or less perfect [illegible] Ebstein has collected twenty cases of [illegible] least two were simply hyperæsthetic [illegible] the paralysis preceded the erup[illegible] which most commonly suffers, and [illegible] herpes of the trigeminal or occipito[illegible] recorded by Lesser, frontal herpes was [illegible] fourth cranial nerve, the inflammation [illegible] from one nerve to the other in the [illegible]

[illegible] need be said is that the severity and [illegible] vary directly with the age of the [illegible]

[illegible] of herpes does not fall within the [illegible] however, be mentioned that the pain is [illegible] failing which cocaine may be tried.

[illegible] des Hôpitaux,' 1876.

PART III.

DISEASES OF THE SPINAL CORD.

INTRODUCTION.

ANATOMY OF THE SPINAL CORD.[*]

THE spinal cord, it will be remembered, is much shorter than the spinal canal, reaching only to the second lumbar vertebra. Hence the nerve-roots descend to their foramina of exit. The lower they arise, the longer is their intra-spinal course. All those below the second lumbar pair leave the canal below the lowest portion of the cord. It is customary to speak of the portion of the spinal cord from which each pair of nerves arise as the corresponding "segment" of the cord. The segments are longest in the dorsal region, and shortest in the lumbar enlargement. They are also called "metameres."

The only parts of the spinal column that we can usually feel are the vertebral spines. Many of these are not on a level with the bodies of their vertebræ. It is important, therefore, to know the relation of the spines to the bodies of the several vertebræ, and of these to the origin of the nerves. These relations are shown in the accompanying figure (Fig. 64).

Of the *Membranes*, the pia mater closely invests the surface, and is continuous with the tracts of connective tissue that pass within the substance of the spinal cord. It is also prolonged along the nerve-roots as their sheaths. The arachnoid forms a much less close investment. The dura mater is not in contact with the bones as it is in the cranium, but a layer of fat and a plexus of large veins intervene between the two. It thus forms a loose sheath for the cord, and variations in the amount of blood in the plexuses outside it permit corresponding (but inverse) variations of the amount of cerebro-spinal fluid within it. An extension of the dura mater passes along each nerve-root, and blends with its sheath.

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[*] The following outline of the anatomy of the spinal cord is intended merely to place before the reader those points that are essential, or likely to become so, for understanding the functions of the organ and the symptoms of its disease. It does not profess to be exhaustive even in outline, and is designedly kept as free as possible from whatever, in either the substance or terminology of recent science, does not come within the scope of those objects. Many of the results reached by modern investigators are mutually incompatible, and where a choice has been necessary the observers have been followed who possess the greatest authority and whose conclusions best agree with facts previously ascertained.

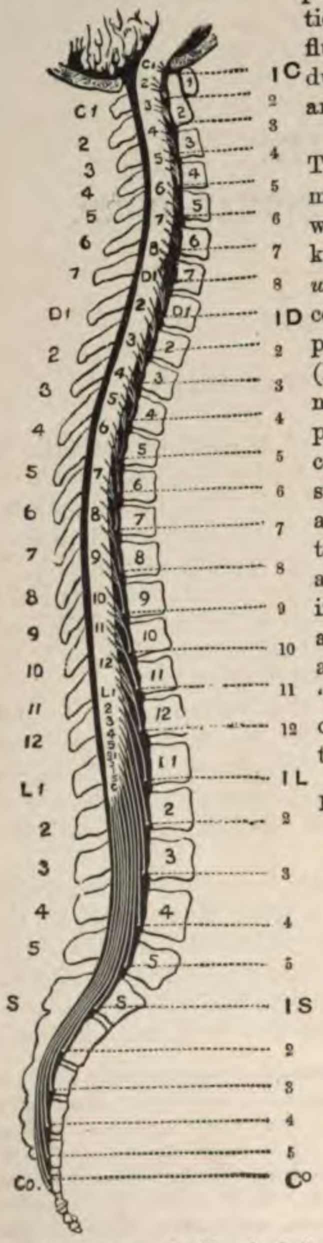

FIG. 64.—Diagram (framed from an original investigation) showing the relation of the vertebral spines to their bodies and to the origin of the several nerve-roots. It will be seen that the ends of the vertebral spines are opposite the middle of their own bodies only in the lumbar region; they correspond to the lower edge of their own bodies in the cervical and the first two dorsal vertebræ, and to the upper part of the body below in the rest of the dorsal region. Each cervical spine is nearly opposite the lower roots of the nerve below; the vertebra prominens is opposite the first dorsal roots, and from the 3rd to the 10th dorsal the spines correspond to the second root below; the 11th spine corresponds to the 1st and 2nd lumbar nerves, the 12th to the 3rd, 4th, and 5th; the 1st lumbar to the 1st, 2nd, and 3rd sacral nerves, while the tip of the cord is opposite the upper part of the 2nd lumbar.

STRUCTURE OF THE SPINAL CORD.—The general form of the cord, the enlargements it presents, and its constitution of white and grey substance, are too well known to need description here. The *white substance* surrounds the grey, except at the two points at which the posterior horns come to the surface (Fig. 65). It consists of medullated nerve-fibres, chiefly longitudinal. The posterior cornua isolate the posterior columns from the rest of the white substance. These columns are separated by a "posterior median septum" of connective tissue, and a little distance from this another incomplete septum, "posterior intermediate septum," corresponding to a depression on the surface, marks off a portion next the median septum, the "postero-median column," or "column of Goll," from the part next the posterior horn, "postero-external column," or "column of Burdach," the part of which next the cornu is called the "posterior root-zone," because many fibres of the posterior root pass through it. The rest of the white substance is divided, in the middle line in front, by the anterior median fissure, down which the pia mater and blood-vessels pass, and at the bottom of which is the anterior or white commissure. Between the anterior median fissure and the posterior cornu the white substance is continuous and undivided, extending round the front and side of the cord. It is artificially divided into an anterior and a lateral column, the line of division being the outermost of the anterior nerve-roots, which pass through the front of the cord; but there is no corresponding distinction of structure, and hence it is often termed the "antero-lateral column."

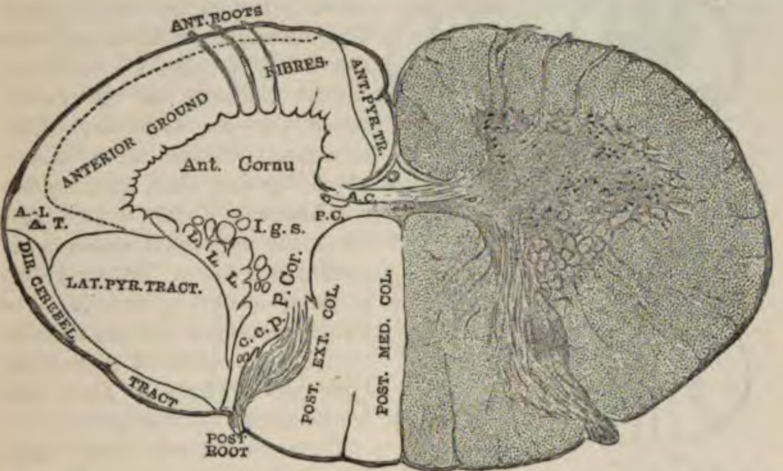

FIG. 65.—Diagram of a section of the spinal cord in the cervical region. A. C., anterior commissure; P. C., posterior commissure; I. g. s., intermediate grey substance; P. Cor., posterior cornu; C. C. P., caput cornu posterioris; L. L. L., lateral limiting layer; A.-L. A. T., antero-lateral ascending tract, which extends along the periphery of the cord.

The white substance varies in amount in different parts of the cord, but, as a whole, lessens gradually from above downwards (see Fig. 66). It is everywhere composed of medullated nerve-fibres, which, however, possess no neurilemma-sheath. In carmine-stained sections the axis-cylinder is seen within each fibre, not always in the centre, and around this are concentric cloudy lines due to the irregular refraction of the white substance.

Between the fibres is a peculiar substance, the "neuroglia," or nerve-cement. It appears to consist of nucleated branched cells ("spider-cells"), whose long, delicate processes form the fibres of the neuroglia. The cells, stellate on section, are found in the larger interspaces between the nerve-fibres. The fibres of the neuroglia branch and interlace, forming a very fine network; some of them are arranged radially; starting centrally from the outer ends of the ciliated epithelial cells which line the cerebro-spinal canal, they diverge, branching as they go towards the surface of the cord. This neuroglia is developed from the same (epithelial) embryological elements as the nerve-structures—an important fact, because it enables us to understand the fact that, in defective development of the nerve-structures, the neuroglial tissue is excessive in amount. But its differentiation in development involves also its chemical nature, since it contains far more of a material like that of hair or horn, and hence called "neurokeratin."

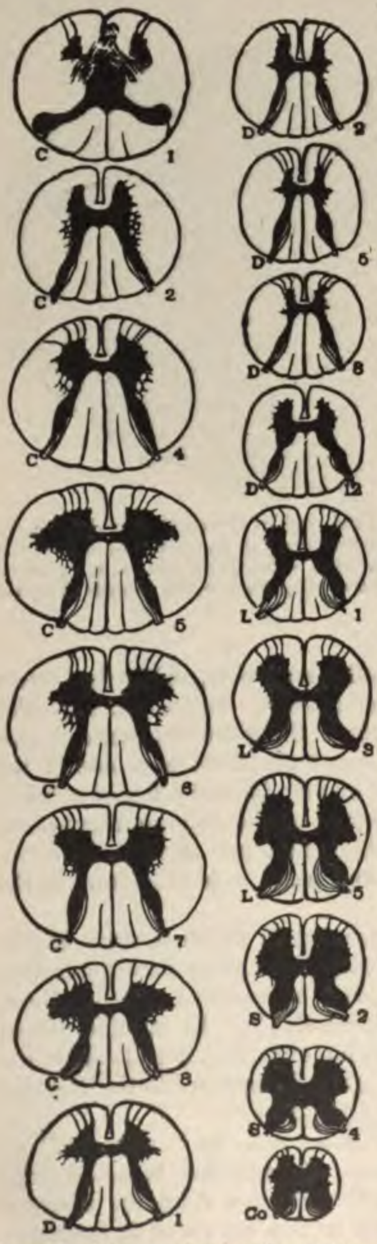

FIG. 66.—Diagram showing the relative size and shape of the cord and grey matter at different levels.

The nerve-fibres, thus connected, lie in a coarser network, formed by branching processes of septa that proceed inwards from the pia mater. In these septa blood-vessels run, and some are occupied by nerve-fibres that have a horizontal course. The septa consist of fine fibres of ordinary connective tissue from the pia mater, and of neuroglial material, which forms also a layer between the membrane and the nerve-fibres in the intervals between the septa. This layer varies considerably in thickness. Many of the septa pass through the white substance to join projections from the grey substance, which cause the irregularity of the outline of the latter. These processes, outside the neck of the posterior cornu, are broad, and join each other so as to form a sort of network, the meshes of which enclose columns of nerve-fibres. This is called the "lateral reticular formation" (Fig. 65, right side; also 66, c 2—8). The substance of the horn behind the caput is sometimes broken up in the same way—the "posterior reticular formation."

The *grey substance* varies in shape and size in different parts of the cord, being largest in the cervical and lumbar swellings, and corresponding to the number and size of the nerve-roots given off. The variations in shape and size are indicated in the accompanying figure (66), and will be readily understood by an examination of this. The division into anterior and posterior horns or cornua is familiar. The part on each side that intervenes between the two horns may be conveniently termed the "intermediate grey substance" (I. g. s. in Fig. 65). In the dorsal region a projection from this extends into the lateral column (Fig. 66, D 2, 5, and 8), and has been termed the "lateral horn"). It is customary to divide the grey matter into two varieties, "spongy" and "gelatinous." The latter forms a cap on the posterior horn, and a layer immediately around the central canal, and is named on account of its naked-eye aspect. It consists largely of neuroglia, traversed, however, by nerve-fibres, and with numerous nerve-cells scattered through it. The spongy substance, which forms the rest of the two cornua, consists mainly of an excessively fine felty network of very narrow medullated nerve-fibres, naked axis-cylinders, and fine nerve-fibrillæ which arise, in part at least, from the dendrites of the nerve-cells, and in part by a division of the axis-cylinders of the nerve-fibres. These structures are embedded in a supporting neuroglia. Through it course many larger medullated nerve-fibres, passing to or from the white columns and nerve-roots. In it also lie many nerve-cells of various sizes. Those in the anterior cornu are, for the most part, large "ganglion-cells;" each contains a large nucleus, and usually a mass of pigment, and

FIG. 67.

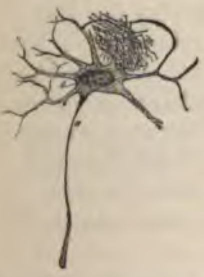

FIG. 68.

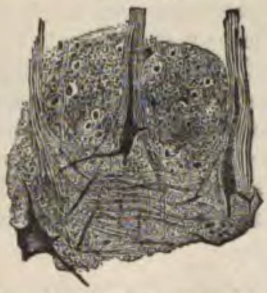

FIG. 69.

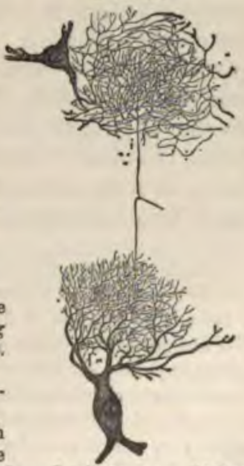

FIG. 67.—A nerve-cell from the anterior cornu of the spinal cord of man: *a*, unbranched process becoming the axis-cylinder of a nerve-fibre; *b*, pigment mass. (After Gerlach.)

FIG. 68.—Nerve-cells of the anterior cornu sending a process into the anterior root. (After Henle.)

FIG. 69.—A fine nerve-fibre dividing into two parts, each of which joins the plexus of fibrillæ formed by the branching processes of a nerve-cell. From the spinal cord of an ox. (After Gerlach.)

sends out several processes. One process, the axon, is continuous with the axis-cylinder of a nerve-fibre (Fig. 67), and can be sometimes traced into a fibre of an anterior root (Fig. 68). The other processes, or dendrons, after a longer or shorter course divide and subdivide, their ramifications ending in minute knobs in the sponge-like matrix of the grey substance. In this they are probably brought into close contiguity with the terminal ramifications of other neurons (Fig. 69). These cells lie in groups, between and through which course tracts of fibres, chiefly from the anterior roots. Other fibres come from the anterior commissure, white columns, and posterior horn. The axons of these cells are certainly motor in nature, and become the motor fibres of the spinal nerves. Similar cells are also found in varying numbers at the anterior periphery of the cord, and even among the fibres of the already descending anterior roots.[*] Birge, in a careful investigation, found that in each segment of the spinal cord of the frog the number of nerve-cells in the anterior cornu nearly corresponds to the number of fibres of the anterior roots arising from that segment. Each cell is traversed by many nerve-fibrillæ, such as are discerned in a cell-process or in the axis-cylinder of a nerve-fibre. These cross and intermingle in the middle of the cell, but pass along the margin from one process to another. By this arrangement impulses entering the cell by one process may leave it by many.[†]

The arrangement of the nerve-cells of the anterior cornu is of some practical importance, on account of the frequency with which this part is the seat of limited lesions. They form, as already stated, certain groups, but these groups vary in different regions of the cord, partly under the influence of the shape of the horn, partly irrespective of this; and they even vary in parts of the cord that are near together.

They are influenced by the course of the tracts of fibres of the anterior roots, which may pass through a group and break it up into smaller groups, although in a neighbouring section it is undivided (see Fig. 70, the cervical groups). Hence very different descriptions have been given of these groups, and the process of distinction has sometimes been carried too far. The groups that can most readily be recognised are the following:—In the inner anterior angle of the cornu is a small group, the *inner* or *median group*. This is one of the smallest, and is absent in some parts of the cord, especially in the lumbar region. A much larger group lies near the anterior edge of the horn, in the middle, or a little to the outer side of the middle, the *anterior group*. External to this, in the outer extremity of the front part of the horn, is another group, the *antero-lateral group*. These two groups are often blended, so that the anterior group cannot be separately distinguished, as in the right-hand mid-cervical figure. A fourth group, usually the largest, lies in the outermost part of the horn, behind its front, usually in the posterior outer angle; it may extend inwards, halfway across the horn. It is called the *external* or *postero-lateral group* (P.-L., Fig. 70). These three are the most important groups.

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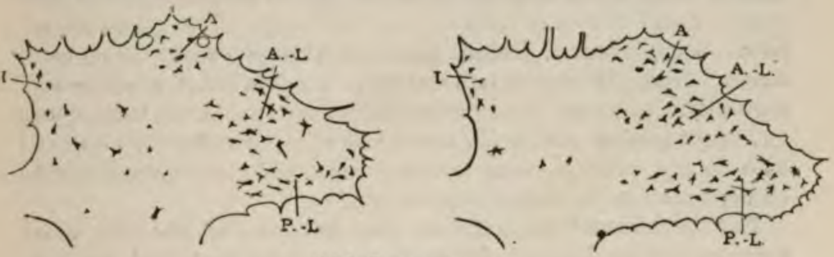
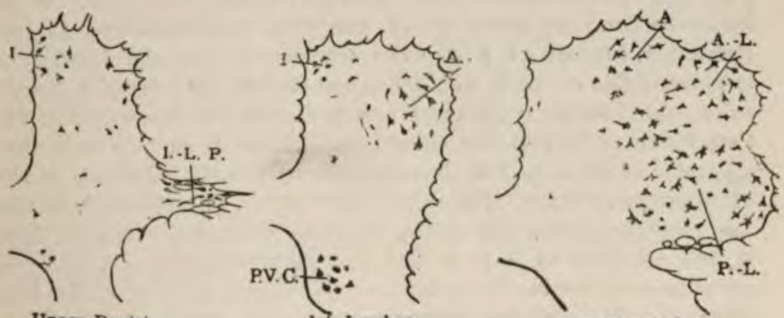
[*] Hoche, 'Neurol. Centralbl.,' 1891.

[†] Kronthal, 'Neur. Centralbl.,' 1890, p. 40. Impulses may thus be diffused, even if there is no junction of fibrillæ or passage of impulses from one fibril to another. This arrangement provides a mechanism by which the nerve-cells may conduct and diffuse impulses, but such a mechanism does not exclude (as has been assumed) the reinforcement or origination of such impulses, and their discovery leaves untouched the facts which demonstrate the trophic influence of the cells, and the manner in which they arrest secondary degeneration. These functions are indicated by facts of a totally different nature. Kronthal's observations are of great importance, and appear trustworthy.



Mid-Cervical



Upper Dorsal 1st. Lumbar. Mid-Lumbar.

FIG. 70.—Diagrams of the groups of nerve-cells in the anterior cornu. Groups:—I., inner or medial; A., anterior; A.-L., antero-lateral; P.-L., postero-lateral; I. L. P., intermediate lateral process; P. V. C., posterior vesicular column or tract. The two mid-cervical sections are only a few millimetres apart, and show how the anterior group, separate in the one, may be blended with the antero-lateral group in a neighbouring part of the cord.

There is in some parts also a *central group*, occupying nearly the centre of the horn. In the small cornu of the dorsal region often no well-defined groups can be made out, but when any can be recognised, they are generally the anterior and external.

Similar cells, usually smaller in size and isolated, are scattered through the intermediate grey matter, and a group of cells occupies a projection outwards into the lateral column in the lower cervical and upper dorsal regions. It was termed by Lockhart Clarke the *intermedio-lateral tract*, but is better designated the *intermediate process* (or *lateral horn*), and the cells the *intermediate group* (I. L. P., Fig. 70).

Nerve-cells also lie in the grey matter of the reticular formations, lateral and posterior, and are sometimes described as special groups. Many isolated cells, medium and small in size, also lie in the posterior horn. These are probably connected with some of the fibres of the posterior roots. They extend back into the caput cornu posterioris.*

Throughout the dorsal and in the upper lumbar cord a group of nerve-cells lies in the inner part of the neck of the posterior horn, the *posterior vesicular column* of Lockhart Clarke (sometimes called "Clarke's column") (P. V. C., Fig. 70; see also Fig. 80). Most of these cells are fusiform in shape, but are placed vertically, so that they appear round or oval in transverse section, and their processes are not seen. Some, however, present processes that run forwards or backwards. Nerve-fibres pass into and by it, which we shall consider subsequently. Although this tract is chiefly developed in the lower dorsal and upper lumbar cord, a few nerve-cells of similar character are met with in the same position in other parts, and are occasionally sufficiently numerous to form a small group.

The "gelatinous" grey matter, *grey substance of Rolando*, which forms a cap on the posterior horn, *caput cornu posterioris* (C. C. P., Fig. 65), differs considerably from the spongy substance. It has been described as made up of granules, but, according to later researches, consists of a peculiar translucent material forming an excessively fine network, which, like the rest of the neuroglia, behaves to reagents like horny material. In the embryo it is continuous with that which surrounds the central canal,† but from this it becomes separated in the course of development. Its basis substance is thus of neuroglial nature. Numerous nerve-fibres pass through it from the posterior roots, and these, coursing forwards, seem to divide it into columns, as it is viewed in transverse section. A few small ganglion-cells lie in this gelatinous substance, chiefly on the inner side; it also contains many minute cells, some of which are nerve-cells, while others belong to the neuroglial tissue. From the nerve-cells axons have been traced into the posterior column and horn, the posterior root-zone and the lateral column.‡ Bundles of vertical fibres are also seen in transverse section, and these are often numerous just above the caput, especially on the inner side of the horn. Most of these are fibres of the posterior nerve-roots that have a vertical course for a short distance. In Fig. 80 the axis-cylinders of these fibres are seen to be changing from the horizontal to the vertical direction, and their relation to the posterior root-fibres can be distinctly traced. The hinder part of the gelatinous substance is less dense in structure than the rest, and is sometimes distinguished from it as the "spongy zone."* The gelatinous substance contains many vessels, running for the most part vertically.

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* It is generally believed that their branching processes enter into the plexus of fibrillæ in the grey matter, and thus bring these cells into relation with those of the anterior cornua, and also with fibres that cross by the posterior commissure and form the ascending sensory path to the brain.

† Cf. Corning, 'Arch. f. mikros. Anat.,' xxxi, 1888.

‡ Starr, 'Atlas of Nerve-cells,' p. 28; Lenhossek, 'Der feinere Bau des Nervensystems,' pp. 120 *et seq.*

White Substance.—We may now examine, in greater detail, the constitution of the white columns of the cord, and the probable course and function of the fibres they contain. Their analysis has been greatly aided by three facts,—continuity of degeneration, difference in time of development, and the variation in the size of the fibres of the different tracts. 1. It was discovered by Türck that in certain tracts in the white substance the nerve-fibres undergo secondary degeneration when separated from their cells of origin, a degeneration analogous to that which, as we have already seen, occurs in the nerve-fibres outside the cord. This mode of investigation has of late acquired a fresh impetus from the new means of staining degenerated nerve-fibres devised by Marchi. 2. In the developing cord the fibres of different tracts acquire their white substance at different periods, and the study of these differences (first by Flechsig and later by Bechterew and others) has not only confirmed, but also extended, the differential indications afforded by disease. It enables us to make a distinction between fibres that do not degenerate separately, or not through a sufficiently long tract to enable us to distinguish them. The groups into which this method enables us to divide the fibres, and the order in which the medullation takes place, are as follows:

(1) Anterior and posterior root-fibres.

$$\left.\begin{array}{l}(2)\\(3)\\(4)\end{array}\right\}\text{Ground-fibres of the}\left\{\begin{array}{l}\text{inner anterior column.}\\\text{postero-external column.}\\\text{outer anterior and lateral column.}\end{array}\right.$$

(5) Lateral limiting layer and antero-lateral ascending tract.

(6) Postero-median column.

(7) Direct cerebellar tract.

(8) Lateral and anterior pyramidal tracts (which in man are first medullated at the time of birth).† It may be stated as a general rule that the shorter the course of a tract within the central nervous system, the earlier do its fibres acquire their medullary sheaths.

3. The size of the nerve-fibres varies in different tracts, but the full significance of the variations has still to be ascertained. Even in the same tract of the cord considerable variations are met with, which probably correspond with differences in function. When a tract degenerates, it is common for the degeneration to be incomplete; fibres remain intact among those that perish: this indicates a difference in function, and with it the difference in size usually coincides. It is also possible that some of the small fibres seen among larger ones may be due to the division of the latter (Sherrington); but such division

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* So that in some descriptions the posterior horn is divided into (1) spongy substance of the horn itself, (2) gelatinous zone of the "caput," and (3) spongy zone at the tip of the horn. See Lissauer, 'Arch. f. Psych.,' 1886.

† See Obersteiner, loc. cit., p. 192.

has not been actually demonstrated, and is probably not frequent. The variation in size is from 1 to 25 μ ($\frac{1}{25000}$ to $\frac{1}{1000}$ inch). The variations are especially great in the fibres of the pyramidal tract, and it is in these that division has been thought to occur. Those of the direct cerebellar tract are uniformly large, while most of those that constitute the postero-median column are small.

It will facilitate the detailed study of the tracts if we consider first those that undergo secondary degeneration for a considerable distance, as our knowledge regarding these is the most complete.

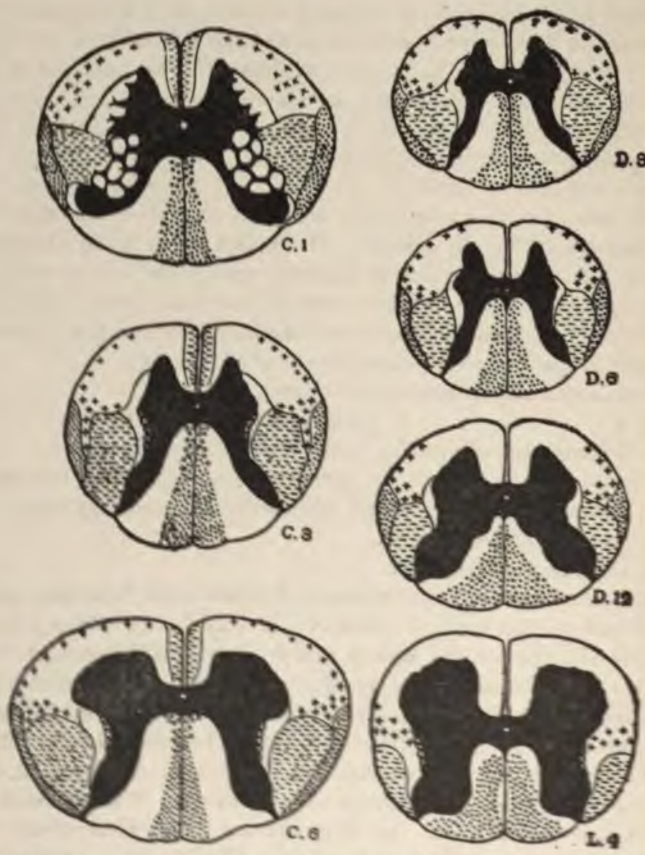

FIG. 71.—Diagram of the elements of the white substance at various levels of the spinal cord. (Modified from Flechsig.) The pyramidal tracts are shaded by short horizontal lines; the direct cerebellar tract by dots; the posterior median column by dots, as it degenerates in disease of the lowest part of the cord or of the nerves of the cauda equina; the small circles in the forepart of this column indicate the area which degenerates in disease of the cervical enlargement. The antero-lateral ascending tract is shown by crosses. The anterior ground-fibres, lateral limiting layer, and postero-external column are left white. Fig. 66 should be compared with this.

These degenerations are both ascending and descending. Only one important set of fibres degenerates downwards through a considerable extent—those that continue through the cord the anterior pyramids of the medulla. They commence still higher, in the motor region of the cortex of the cerebral hemisphere, passing thence by the "internal capsule" between the two parts of the corpus striatum, &c., to the crus and pons; they are the chief, perhaps the sole conductors of voluntary impulses. They are termed the *pyramidal tracts* because they constitute the anterior pyramids of the medulla. They are shaded by short horizontal lines in Fig 71; compare also Fig. 65.

At the decussation of the pyramids about three quarters of the fibres usually cross to the other side. These pass down the cord in the lateral column, and constitute the *lateral* (or *crossed*) *pyramidal tract*. Those which do not decussate, pass down their own side of the cord, in the inner part of the anterior column, at the side of the anterior median fissure, and constitute the *anterior* (or *direct*) *pyramidal tract*. Flechsig has found that the decussation is subject to many variations. In the majority of cases the decussating fibres are between 70 and 80 per cent. of the whole. Sometimes they constitute a still larger proportion, and in one case (of 60 examined) all the fibres crossed. Sometimes fewer fibres decussate, only one half or even less than half; in one case 35 per cent., in another only 10. No case has yet been met with in which no fibres crossed.* A considerable number of fibres decussate above the medulla, terminating around the cells which give origin to the motor cranial nerves.

The *lateral pyramidal tract* occupies the posterior half of the lateral column, outside the posterior cornu. It extends down to the end of the cord, even when it is originally small. Hence those fibres that do not cross at the medulla must do so lower down in the cord. Through the greater part of the cervical and dorsal regions this lateral tract is separated from the surface by a narrow layer of fibres, the *direct cerebellar tract*. In the upper part of the cervical region (third cervical segment) this tract lies farther forwards, so that the pyramidal tract comes up to the surface close to the posterior cornu (Fig. 71, C. 3); and here, if the tract is small, there may be a depression on the surface. In the lower part of the dorsal cord there is a similar movement forwards of the cerebellar tract, so that the pyramidal tract again comes in contact with the surface posteriorly (Fig. 71, D. 12), and, as the cerebellar tract ceases at the first lumbar nerve, the pyramidal tract extends up to the surface throughout the lumbar enlargement (Fig. 71, L. 4). The inner side of the tract is in contact with the hinder part of the posterior cornu, near the surface, throughout the entire length of the cord, but further forwards it is separated from the neck of the cornu and intermediate grey substance by a layer of nerve-fibres, termed by Flechsig the *lateral limiting layer* (L. L. L., Fig. 65). Among the fibres of the pyramidal tract, in the dorsal and cervical regions, degeneration reveals a few other fibres that belong chiefly to the cerebellar tract. The lateral pyramidal tract diminishes in size from above downwards as its fibres end by entering the grey matter; those that extend lowest (for the leg) occupy the posterior part of the tract in the cervical region. The fibres curve inwards along the septa that extend from the grey matter into the lateral column, and they enter the grey matter between the anterior and posterior cornua. They pass inwards and forwards in the anterior cornu, and are then lost in the complicated structure of the grey matter. Their probable termination will be considered presently.

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* In the 'Neur. Cent.,' 1898, p. 202, Dr. Philip Zenner describes a case of tumour of the left side of the brain in which the paralysis was on the left side of the body. The post-mortem examination showed that there was "absence of the pyramidal decussation." It is not stated whether Marchi's method was used in the examination.

The *anterior* or *direct pyramidal tract* (called also the "column of Türck") descends the cord in the inner part of the anterior column, adjacent to the anterior median fissure. Its exact form and size vary: usually it bounds the fissure on each side; sometimes it forms only a small tract at the posterior part of the fissure; when large, it not only reaches forward to the front of the cord, but may cause a prominence on the surface bounded by a distinct sulcus. Its extent down the cord probably varies according to its size. It steadily lessens in area, and, if originally small, may not extend beyond the middle of the cervical enlargement; if originally large, it may be traced as far as the lumbar enlargement and even into the cord at the level of the fourth pair of sacral nerve-roots[*] (Fig. 72); when bearing the usual proportion to the lateral tract, it ceases about the mid-dorsal region. Most of the fibres pass through the anterior commissure to the opposite side, either entering at once the grey matter, or passing first to the lateral tract.[†] The latter is probably the course of most of the fibres when the pyramidal decussation is small. Whether any fibres end in the grey matter on the same side of the cord we do not know.

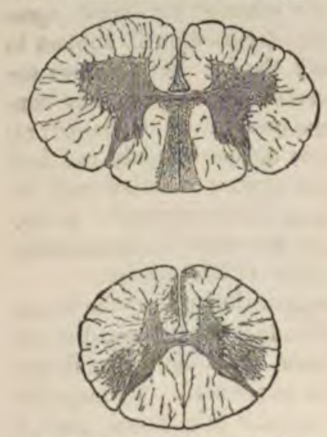

FIG. 72.—Ascending and descending degenerations from myelitis of the mid-dorsal region; the upper section is from the lower cervical, and shows the ascending degeneration of the post.-median column spreading out against the commissure. The other figure is from the lower dorsal, and shows degeneration of the pyramidal tracts, anterior and lateral.

Disease of the brain destroying the motor cortex, or the fibres which descend from it to the pyramids, causes descending degeneration usually limited (at least in conspicuous degree) to the related pyramidal tracts,—anterior on the same side, lateral on the opposite side (Fig. 73). But in many cases slight degeneration has been also found in the lateral tract on the same side, extending into the lumbar region. Hence it is probable that some fibres of each anterior pyramid find their way to the lateral tract on the same side, and descend to the corresponding limbs, especially to the leg. This degeneration of the lateral tract on the same side as the lesion is sometimes very marked just below the decussation of the pyramids, and is therefore not due to any lower re-decussation in the cord.* Muratoff has shown, by extirpation of the cortical areas for the extremities, that a bundle from the bulbar pyramid descends into the lateral pyramidal tract of the same side. This has been confirmed by Rothmann and also by Mellus and by Sherrington, who formerly held a different view.† That there is such a physiological relation is clearly indicated by clinical facts. We shall afterwards see that there is abundant evidence that each hemisphere of the brain is connected with both legs, although chiefly with that of the opposite side. There is also a similar and even more equal connection with the trunk muscles, and a slighter connection with certain muscles of the arm. Such muscles invariably regain some power on the paralysed side, even with a complete inter-

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[*] Dejerine and Thomas, 'Compt. Rend.,' 1896, p. 157. Russell traces it to the 5th segment.

[†] Although this is probable there is no definite evidence of it except in the sacral region (Russell, 'Brain,' 1898).

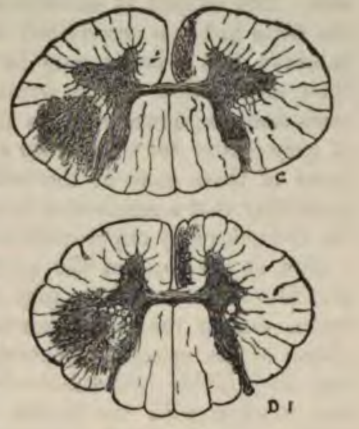

FIG. 73.—Descending degeneration of the pyramidal tracts in a case of hemiplegia from disease of the right cerebral hemisphere. (From sections prepared by Dr. Mott.)

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* Pitres, 'Arch. de Physiologie,' 1874; Hadden and Sherrington, 'Brain,' Jan., 1886. An illustration of this bilateral degeneration will be found in the section on Diseases of the Brain, Vol. II.

† Muratoff, 'Neur. Centralbl.,' 1893; Mellus, 'Proc. Royal Society,' 1894; Sher-

supplies to the motor path, and they are weakened on the unpa side. The subject is considered further in Vol. II. On the the facts hitherto ascertained suggest that the arrangements whereby this relation vary in different persons, just as do amount of decussation of the pyramids. The fibres probably in some continue downwards on the same side until they reach th of grey matter for which they are destined.* In other cases m many of them cross at the bulbar decussation Lastly, there possibility that a connection between the motor cells or structu the two sides may supplement the decussation.†

How do the fibres of the pyramidal tract end? They seem pass into the intermediate grey matter, and forwards into the an cornu among the motor nerve-cells. There they are lost i plexus of the spongy substance. There is strong indirect evi that they divide and subdivide, and that their ramifications into relation with the plexus of nerve-fibrillæ constituted b dividing processes of the ganglion-cells. The evidence of t twofold. As far as is at present known, only one process ganglion-cell becomes an axon; the others divide. The axon p into an anterior root; it can be seen to do so in the case of ganglion-cells (Fig. 68), and, as we have seen, the number of gang cells and anterior root-fibres is nearly the same in the frog, therefore, presumably, in man also. Since some root-fibres are motor, the number of motor cells may safely be assumed to least not smaller than that of the motor fibres; thus each fibre have at least one cell related to it. If so, since the pyramidal fi are certainly connected with the ganglion-cells, the connec can only be by the dendrons or ramifying processes of the and this involves a division of the nerve-fibre. The dividing isolated by Gerlach, and shown in Fig. 69, may have been a bra of a pyramidal fibre. The second point in evidence of divisio the fact that the nerve-cells and root-fibres must be many ti more numerous than the fibres of the pyramidal tract.‡ But (or at least most) of the motor root-fibres may be stimulated thro the pyramidal fibres, and therefore each of the latter must be nected with several ganglion-cells. Physiological considerations gest the same conclusion, which is fully confirmed by the new hi

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rington, 'Lancet,' 1894, vol. i; Rothmann, 'Neur. Centralbl.,' 1896; Redlich, il 1897. Their results, obtained by the method of degeneration, have been fur borne out by the ingenious experiments of Wertheimer and Liepage ('Arch Physiol.,' 1897).

* Dejerine and Thomas have traced homolateral fibres as low as the 4th sa root ('Compt. Rend.,' loc. cit.), Russell to 5th root ('Brain,' loc. cit.).

† Broadbent, Hadden. In a unilateral lesion of the cord the paralysed foot more with the other. Such a connection of nuclei was originally suggested Broadbent to explain the association of bilateral movements.

‡ The total area of the anterior roots of the cord is probably at least five t as great as that of the pyramidal tracts in the upper cervical region.

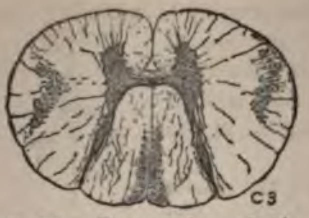

FIG. 75. — Degeneration of the antero-lateral ascending tract in a case of locomotor ataxy (q. v. figure), showing its position in the upper cervical region, where the direct cerebellar tract passes outside it into the restiform body.

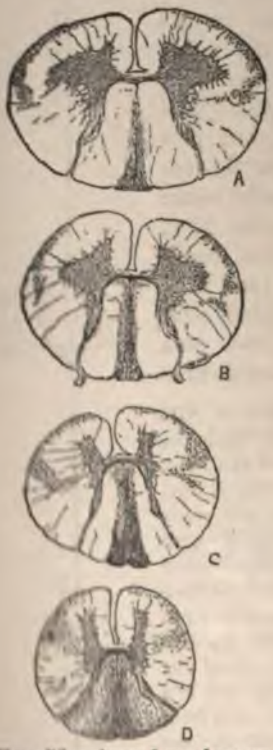

FIG. 76.—Ascending degeneration in the postero-median column and antero-lateral ascending tract. The cord was crushed at the first lumbar segment.

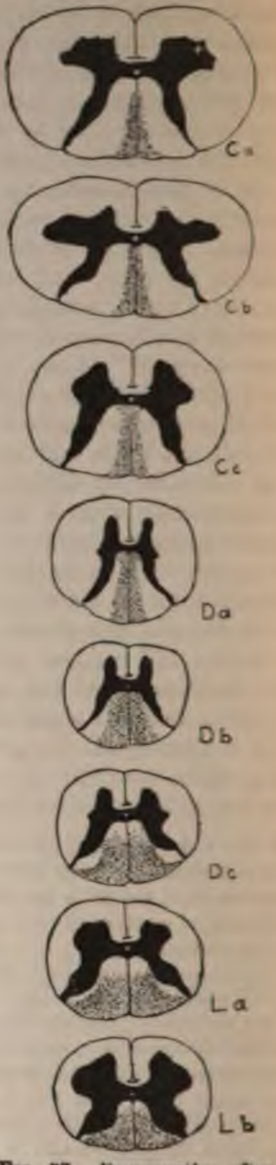

FIG. 77.—Degeneration after injury to the cauda equina. C a, b, c, upper middle and lower cervical; D a, upper; b, c, lower dorsal; L a, upper; and L b, middle lumbar region. (After Schultze.)

of excitable muscle-tissue. It will be found that this conception of the [illegible] conduces to clearer ideas of many facts of disease, and it is important to grasp it firmly. We shall see, for instance, that diseases involving any part of a neuron produce similar effects, however diverse their nature; while there is a fundamental difference between the effects of disease of the two neurons.[*]

Fibres that degenerate downwards through the greater part of the cord are also scattered through the antero-lateral column, in a zone that extends beneath the surface between the direct pyramidal tract and the direct cerebellar tract. Some are mingled with the fibres of the ascending antero-lateral tract presently to be described; others lie beneath this. It is possible that they really belong to the crossed pyramidal tract. They have been termed by Foster the *descending antero-lateral tract.*[†]

Bechterew has pointed out that the crossed pyramidal tracts as mapped out by the degeneration and embryological methods do not correspond. In other words, the area of non-myelinated nerve-fibres found at birth is much greater than that degenerating as the result of section of the pyramidal tract high up. Boyce and Sakowitsch have shown independently that the axons of the remaining area—which is situated at the anterior end of the crossed pyramidal tract—descend from the posterior corpora quadrigemina.[‡]

Fibres degenerate upwards in both the lateral and the posterior columns. We may consider the latter first.

Each *posterior column*, as we have seen, is divided by the imperfect "intermediate septum" into two parts,—a narrow "postero-median column," and a wider "postero-external column" (Fig. 65). In the lumbar and sacral regions it is not possible to separate the postero-external from the postero-median column, as can be done in the dorsal and cervical regions. The differentiation is not completed below the level of the eleventh dorsal segment. Secondary degeneration confirms this distinction. If the posterior columns are interrupted anywhere in their course, ascending degeneration results; and this, a short distance above the lesion, is confined to the posterior median column (Figs. 72, 76, 77), in which the degeneration continues upwards to the medulla oblongata, and ends there at the grey matter of the "nucleus gracilis." The fibres of these median columns degenerate in the same way when the lesion is not in the cord, but in the posterior nerve-roots (as of the cauda equina, Fig. 77). Hence it is clear that these fibres are continued upwards from the posterior nerve-roots without interruption. The contrast

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[*] This conception of the motor path was published in its present form (except, of course, that the neuronic nomenclature had not then been introduced) in the first edition of this book in August, 1886. By a curious coincidence nearly the same idea was stated (no doubt independently), and even the same terminology employed, by Dr. v. Renz in the 'Centralblatt f. Nervenkr.,' October 15th, 1886.

[†] Foster, 'Physiology,' 1890, p. 873. See also Hadden and Sherrington, 'Brain,' vol. viii; and Tooth, 'Secondary Degeneration of the Spinal Cord,' 1889. Luciani and Marchi state that this tract is mainly composed of axons descending from the cerebellum, a view which is upheld by Biedl ('Neurol. Centralbl.,' 1895). Russell denies this, and states that these axons are derived mainly from Deiter's nucleus in the medulla, a view with which Mott agrees. Held has shown that fibres derived from the red nucleus pass into the lateral column of the cord ('Archiv f. Anat. u. Physiol.,' 1892).

[‡] See Bechterew, 'Neurol. Centralbl.,' 1897, No. 23, where a tract in the anterior ground bundle descending from the anterior corpora quadrigemina is also described. The term "retrograde degeneration" has been applied by Sottas and others to an ascending change which occurs in certain cases in the pyramidal tracts. This degeneration is slow in developing, and undergoes gradual diminution in intensity and extent as it ascends. A similar condition has been found in the posterior columns after cerebral lesions (Durante, 'Rev. neurologique,' 1898), and in the direct cerebellar tract after a cerebellar lesion (Campbell, 'Brain,' 1897).

FIG. 78.

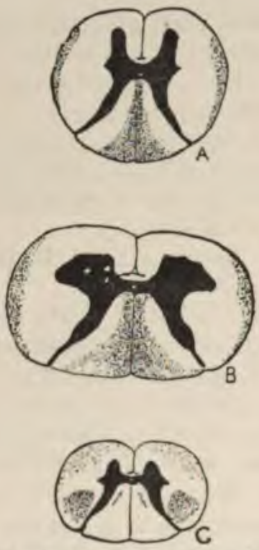

FIG. 79.

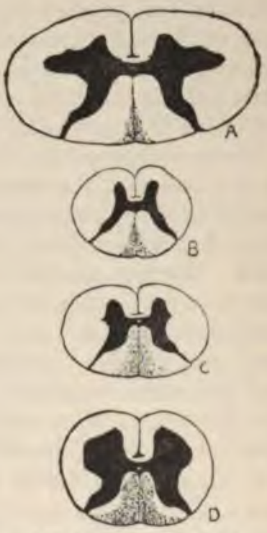

FIG. 78.—Degenerations after a lesion of the cord in the lower part of the cervical region. A, highest cervical; B, middle cervical 1½ cm. above lesion. Degeneration of the posterior median columns, spreading slightly in A and widely in B into the post. ext. col. The nature of the circumferential degeneration is uncertain; it is probably a partial ascending degeneration set up by peripheral myelitis.

C, upper dorsal 2 cm. below lesion. Degeneration of the lateral pyramidal tracts; "comma-shaped" degeneration in front of the post. ext. col.; slight degeneration of the anterior ground-fibres. (Schultze.)

FIG. 79.—Ascending degeneration after injury to the lowest part of the spinal cord and sciatic nerve-roots arising from it. A, cervical; B, lower dorsal; C, junction of dorsal and lumbar regions; D, middle of lumbar enlargement. (After Schultze.)

between these degenerated columns and the undegenerated external columns through the greater part of the cord above the lesion (Fig. 76) is very striking. But just above the lesion the degeneration is not confined to the median columns; it spreads out into the external columns, especially towards the hinder surface, and the more extensively the nearer to the lesion, until close above this the degenerated fibres occupy the whole extent of the posterior column, except a small area close to the posterior cornu. This is due to the fact that the fibres pass to the median column through the hinder part of the external column. It is the same whatever is the seat of the lesion, whether it is in the cervical (Fig. 78), dorsal, or lumbar (Fig. 79) parts of the cord; and if it is in the cauda equina, the degeneration spreads out in the same way in the lumbar enlargement (Fig. 77). This shows clearly that all the way up the cord fibres pass to the median column from the external column, and that these are uncrossed fibres from the posterior roots.[*] As the fibres ascend the cord in the median column, they come to lie posteriorly in proportion as their source is lower. Those from the sciatic nerves, for instance, in the lower cervical cord only occupy the posterior half of the columns (Fig. 79). Those from the whole lumbar enlargement, or whole cauda equina (Fig. 76), reach forward to the commissure in the lower cervical region, spreading out a little near the commissure, where, however, the degeneration is much less dense than it is behind Above the middle of the cervical enlargement the degeneration from a lesion in the dorsal or lumbar regions does not extend forwards beyond the junction of the anterior and middle third of these columns; but if the lesion is in the cervical region the degeneration extends up to the commissure, even at the level of the second cervical segment (Fig. 78, A), showing that the fibres from the cervical roots occupy the anterior portion of these columns. It is now known that most if not all root-fibres divide on entering the posterior columns into ascending and descending branches. With the course of the former we have just been occupied; the latter run but a short way down in the cord and terminate by turning into the posterior cornu and inosculating with the cell-dendrites there found.

It is important to remember that the root-fibres that pass to the median column through the external column do not decussate. This is not, however, the only source of fibres to the median column. Fibres pass to it (1) from the neck of the posterior horn, across the anterior part of the external column, curving backwards (Fig. 80, *x*); many of them extend almost to the postero-median septum. They are "endogenous," *i. e.* they arise from cells of the posterior cornua. In the posterior columns they bifurcate into ascending and descending portions, the former of which may in certain parts of the cord form short but definite tracts, such as the "comma" tract of Schultze[†] and that in the lumbo-sacral region referred to below. These descending fibres terminate in all probability by again turning into the grey matter of the posterior cornua. (2) From the posterior commissure in the middle line (Fig. 80). These are very numerous and conspicuous in the lumbar enlargement. They course backwards in the septum and then pass outwards on each side into the adjacent column. They come from the posterior and lateral columns of the opposite side, decussating in the septum. Some arise from posterior root-fibres, others from cells in the posterior cornua.

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[*] Except in cases in which the cervical roots are affected in which degeneration is restricted to the postero-external column terminating in the cuneate nucleus. Gombault, 'Bull. de la Soc. d'Anatomie,' 1891; Sottas, 'Rev. de Méd,,' 1893; Dejerine and Thomas, 'Compt. Rend.,' 1896; Souquez, 'Compt. Rend.,' 1895; Russell, 'Brain,' 1896.

[†] 'Arch. f. Physiol.,' 1883.

In the lumbo-sacral region there is a narrow tract of fibres close to the posterior median septum, that is apparently of different nature from the rest of the column. It is lenticular on transverse section, and is indicated by the clear area in L. 4, Fig. 71, and faintly in L b, Fig. 77; it is best seen from the third lumbar to the second sacral segments. It is distinguished by a difference in time of development (Flechsig), and by freedom from the secondary degeneration that involves the rest of the column. It has been observed to undergo descending degeneration in a certain number of cases, and its fibres appear to terminate in the posterior horn of the same side. Their origin is uncertain, but they are probably endogenous and not directly connected with the posterior root.* A careful study by Pineles has shown that they fail to degenerate in tabes, and they also escape in the degeneration associated with anæmia.† This tract is possibly identical with Flechsig's "oval field," and is also known as the septo-marginal tract (see p. [illegible]).

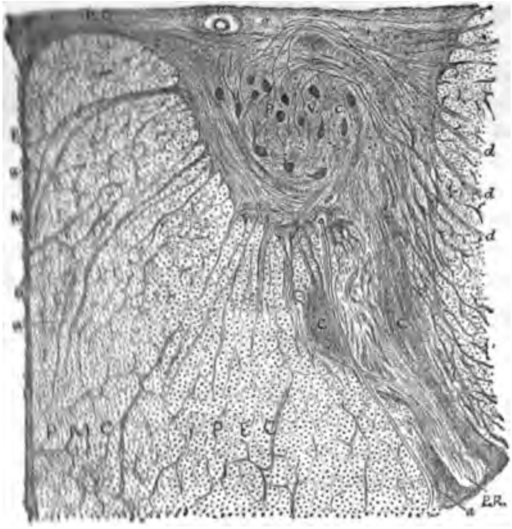

FIG. 80.—Posterior cornu and column at the last dorsal segment. P. M. C., postero-median column; P. E. C., postero-external column; P. M. S., posterior median septum; P. C., posterior commissure; *v*, commissure vein. P. V. C., posterior vesicular column; C. C., caput cornu; P. R., posterior root; *a*, an artery; *d, d, d*, adjacent to a strip of the lateral column, indicate the tracts of fibres passing from the outside and interior of the posterior vesicular column along the septa of the lateral column, to form the direct cerebellar tract; *x, x*, tracts of fibres passing from the neck of the horn, near the post. vesic. col., to the post-median column.

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* See Hoche, 'Neurol. Centralbl.,' 1896; Bruce and Muir, 'Brain,' 1896.

† For an illustration of this tract see later, where the degenerations associated with anæmia are referred to.

It is very doubtful whether all the fibres that enter the postero-median columns continue in these to the medulla. Some certainly do, but the upward increase in size of the columns seems to be far too small for the accommodation of all the fibres that seem to pass to them. At the same time we have, at present, no indication of the mode in which fibres leave these columns.

At the medulla oblongata this column becomes filled with nerve-cells, the *post-pyramidal nucleus*, so called because the highest portion of this column has been termed the *posterior pyramid* of the medulla. It is also called the *nucleus gracilis*. The discovery that the fibres of this column are continuous with the nerve-roots invests the post-pyramidal nucleus with considerable importance, since its nerve-cells are the first with which these root-fibres are related, and secondary degeneration hence stops here. The upward degeneration of the median fibres implies upward conduction; their probable function will be considered in the next section. It is now known that not all of them end in the gracile nucleus; some enter into the external arcuate fibre-system, while others run in the restiform body to the cerebellum.

The *postero-external column* ("column of Burdach," containing the "posterior root-zone" of Charcot) consists chiefly of vertical fibres. Many of the posterior root-fibres pass through it, horizontally or obliquely (Fig. 65). These either curve inwards to the posterior horn (some first inclining upwards), or pass obliquely upwards and inwards to the median column and constitute the fibres of this column that are continued from the nerve-root. Across the anterior part fibres also pass, as just described, between the neck of the posterior horn and the median column. The vertical ground-fibres of the external column have apparently only a short course, since they degenerate for only a few centimetres above or below a lesion. The longest descending degeneration is of a "comma-shaped tract," in the middle of the anterior third of the column in the dorsal region, which may degenerate downwards for eight or ten segments* (Fig. 78). These vertical fibres, of short course, may connect the grey matter of the posterior horn at different (but adjacent) levels; this is rendered probable by the fact that they degenerate in transverse lesions of the cord but not in those of the posterior roots.† This column is larger in the swellings than it is in the dorsal region, chiefly in consequence of the larger number of root-fibres that pass through it. Above, it also ends in a grey nucleus, the *postero-external* nucleus or *cuneate* nucleus.

Both these posterior nuclei are connected directly with the cortex of the opposite hemisphere, and chiefly with the ascending parietal convolution. Fibres also pass from them to the cerebellar hemisphere

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* Hoche, 'Arch. f. Psych. u. Nervenkrank.,' xxvii.

† Gombault and Philippe, 'Sem. Méd.,' 1894. This is only true of experimental division of the posterior roots, not of two cases described by Dejerine and Thomas ('Compt. Rend.,' 1896), and Nageotte ('Rev. Neurologique,' 1895), in which the degeneration was found to extend down the cord. See also Russell ('Brain,' Summer, 1898).

of their own side, which is connected with the same region of the opposite cerebral cortex, probably through the red nucleus and optic thalamus.[*] The two nuclei (of both sides) are also found (by developmental researches) to send fibres through to the inferior cerebellar peduncle,[†] and fibres pass from the posterior median nucleus to the middle lobe of the cerebellum on the same side,[‡] a fact of considerable significance. Two other tracts of endogenous fibres, *i. e.* fibres not derived from the roots but from cells in the grey matter of the cord itself, have been described in the posterior columns—one by Marie, the *cornu-commissural tract*, in close relation to the posterior commissure throughout the sacral and lumbar regions of the cord; the other, which has been named the *septo-marginal tract* (see p. 218), is in proximity to the septum as high as the eleventh dorsal segment. Some observations would seem to indicate that it may originate as high as the cervical regions.[§] These tracts remain undegenerated in tabes dorsalis, and in the degenerations associated with anæmia (p. 218).

The "*direct or dorsal cerebellar tract*" is another series of fibres that degenerate upwards. It forms a layer at the periphery of the lateral column, outside the pyramidal tract (Fig. 65), but does not extend through the whole length of the cord, ceasing below at the level of the first lumbar nerve (Fig. 71). The anterior part of the tract (as seen in section) does not extend forwards beyond the level of the lateral pyramidal tract, although it was formerly thought to do so because there are other fibres in front of it that also degenerate upwards, and these were not distinguished from those of the tract. But near its upper and lower extremities, at the level of the second cervical nerves, and also in the lowest part of the dorsal region, the tract lies a little anterior to its position in the rest of the cord, and hence at these places the pyramidal tract comes to the surface behind the cerebellar tract, close to the posterior nerve-roots, up to which elsewhere the cerebellar tract extends. The tract increases somewhat in size from below upwards, and hence receives fibres throughout its course, but most of its constituent fibres enter it at the level of the lowest dorsal and first lumbar nerves, *i. e.* at its lowest part. These fibres come from the grey substance, passing through the lateral column, along the septa that cross the pyramidal tract from the grey matter. In the latter these fibres are conspicuous objects at this level (Fig. 80, *d*, *d*), passing transversely and obliquely from the front of the posterior vesicular column. Into this many of them can be traced; others change their direction and become vertical, perhaps passing into the vesicular column at a different level. It is now known that the fibres come from this column; the cerebellar tract is chiefly formed at that part of the cord at which the column is most developed, and the cells of this column have been found atrophied when the cerebellar tract is degenerated.* Moreover modern staining methods have demonstrated the origin of the axons of the direct cerebellar tract from the cells of Clarke's column. It has also been shown that axons pass to this tract from Stilling's sacral nucleus, which is the sacral homologue of Clarke's column. Besides the fibres that are gathered into the compact cerebellar tract, others, apparently belonging to it, ascend among those of the adjacent pyramidal tract.

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[*] See Flechsig and Hosel, 'Neurol. Cent.,' 1890, p. 417.

[†] Darkschewitsch and Freud, ib., 1886, p. 121.

[‡] Bechterew, 'Wjistnik Psych., &c.,' 1886.

[§] A. Bruce, 'Brain,' Autumn, 1897.

The cerebellar tract only degenerates when a lesion of the cord is at, or above, its place of origin, the junction of the dorsal and lumbar regions If the lesion is in the lumbar enlargement, as in the case shown in Fig 76, the tract does not degenerate.

At the level of the first cervical nerve (where the pyramidal tract leaves the lateral column to cross into the anterior pyramid of the medulla) the cerebellar tract lies in front of the "grey substance of Rolando" (formed from the caput cornu posterioris), and passes up, in the restiform body, to the cerebellum. Since it degenerates upwards we must conclude that it also conducts upwards.†

We have seen that the lateral pyramidal tract, although in contact, behind, with the head of the posterior cornu, does not extend quite up to the neck of the horn or to the intermediate grey substance, the two being separated by a "*lateral limiting layer*" of vertical fibres, in part broken up by processes from the grey matter In the upper cervical cord, and also to a less extent in the dorsal cord, this layer extends forwards outside the anterior horn, which, in these parts, is narrow It consists of fine fibres that seem to pass into and out of the grey matter. This course, and the fact that the tract does not degenerate through any considerable extent, suggest that its fibres have but a short course, and connect the grey matter of adjacent regions.

There is a small tract of fibres, all of small size, at the junction of the tip of the posterior horn and lateral column known as Lissauer's tract. The fibres come from the posterior roots; they course upward for a short distance, forming the tract, and then enter the posterior horn.

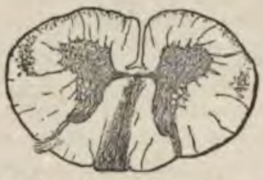

FIG. 81.—Cervical section. Ascending degeneration in post-med. col and ant.-lat. ascending tract, secondary to a lesion in the dorsal region. (From a section prepared by Dr. Mott.)

* *E g.* by Minkowski, 'Deut. Arch. f. kl. Med.,' Bd. xxxiv, p. 433. Slight degeneration of the pyramidal tracts was the only other lesion.

† In the absence of evidence of contrary character, the direction in which fibres conduct must be assumed to be that in which they degenerate. We know of no exceptions to it within the central nervous system. Wherever we can observe the relations this law obtains, except in the peripheral sensory nerves, in which the conditions are conspicuously exceptional. Hence the probability is very great that the correspondence obtains where we have not yet been able to prove it; and to ignore the probability because it is not a certainty is scarcely reasonable, although not uncommon.

The rest of the lateral column, in front of the pyramidal and cerebellar tracts, consists of fibres that vary in size, course, and date of development. Secondary degeneration, however, as I pointed out some years ago,[*] enables us to distinguish an important tract which occupies an irregular area in front of the pyramidal and cerebellar tracts, and degenerates upwards throughout the cord. It extends across the lateral column, as a band which fills up the angle between the pyramidal and cerebellar tracts, and it reaches the surface of the cord in front of the latter tract, nearly on a level with the anterior commissure; it then extends forward in the periphery of the anterior column, almost to the anterior median fissure, and up to the direct pyramidal tract when this exists. I have termed it the *antero-lateral ascending tract* (Figs. 65. 71, 75, 76, and 81). It has often been confounded with the direct cerebellar tract, and hence the impression arose that the latter extends farther forwards than it really does. Posteriorly the tract extends across the lateral column towards the posterior commissure (Fig. 71, L. 4). In the cervical region (ib., C. 3), especially in the upper part, where the direct cerebellar tract extends farther forward, the broadest part of the ascending tract lies just within the anterior part of the cerebellar tract, and extends, like a wedge, between the latter and the pyramidal tract.

This tract has been found by Bechterew to undergo development at a different period from the rest of the lateral column. It apparently constitutes an upward path from the opposite posterior roots, since its degeneration, if unequal on the two sides, is greater on the side opposite to that on which the uncrossed postero-median column is most degenerated. Its fibres do not degenerate when the nerve-roots are divided, and hence probably spring from cells in which the root-fibres end. Fibres apparently pass to it through the whole length of the cord, and these are mingled together, so that the degeneration from a lesion of the lower part of the cord is not dense.[†] The precise form of the tract seems subject to individual variations. Above, its fibres probably end in the cerebellum.[‡]

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[*] 'Diagnosis of Diseases of the Spinal Cord,' first ed., 1879.

[†] Other illustrations of the degeneration of this tract will be found in the section on Locomotor Ataxy. For an example of its degeneration and references to previous observations, see Tooth, 'St. Bartholomew's Hospital Reports,' 1885, p. 137. See also his 'Lectures on Secondary Degeneration in the Spinal Cord,' Lond., 1890.

[‡] Experiments on monkeys show that the antero-lateral ascending tract as here described is distinguishable as high as the origin of the fifth cranial nerve, round which it curves. It then passes backwards on the under surface of the superior cerebellar peduncle to reach the superior vermis. This portion is now known as the ventral cerebellar tract, in contradistinction to the direct or dorsal cerebellar previously mentioned. (See Mott, 'Monatsschrift f. Psych. u. Neurol.,' i, p. 104; and van Gehuchten, 'Anatomie du Système Nerveux,' 2nd ed., p. 764 *bis*.) Mott ('Brain,' 1895) states that the tract can be distinguished after a lesion involving the 3rd, 4th, and 5th lumbar segments in the monkey. Russell ('Brain,' 1898) from pathological evidence confirms this. External to this antero-lateral ascending tract, at the extreme periphery, is a tract of descending fibres—the *efferent or*

Some other fibres are also found in the position of the antero-lateral ascending tract, constituting the "crossed afferent tract" of Edinger. They probably enter from the posterior root and come into connection with cells which give off axis-cylinder processes decussating in the anterior commissure. They pass up near the fillet and end in the corpora quadrigemina, or optic thalamus.[*]

Some fibres of the anterior part of the lateral column are large in size, and curve upwards and downwards into the anterior horn. They must be of short course, and may be fibres of the anterior nerve-roots, which ascend and descend to nerve-cells on a different level from that of the roots by which they leave the cord.

The fibres of the anterior column, excluding the anterior pyramidal tract, are termed by Flechsig the *anterior ground-fibres* (Fig. 65). They are not separable, structurally or by development, from those of the lateral limiting layer. The ground-fibres do not degenerate through any considerable extent of the cord, and some probably connect the anterior cornua at different levels. Moreover some of the fibres pass to the anterior commissure, and thus, by the agency of the anterior columns, a connection may be established between the two anterior cornua at different levels.

The *commissure* of the spinal cord lies between the bottom of the anterior fissure and the posterior columns, and in it alone is there a passage of fibres from one half of the cord to the other. It consists of two parts, an anterior or white, and a posterior or grey commissure. The *white commissure* varies in thickness in different parts of the cord, and is largest in the lumbar region. It is composed of medullated fibres, which cross in the commissure in such a manner that the anterior fibres on each side pass out in the posterior part of the commissure on the other side. The fibres in front pass into the anterior white column, those behind into the grey substance; hence they appear to establish a connection between the anterior column of one side and the anterior cornu of the other; but many of those entering the anterior column merely pass through this to the cornu, and this is probably true also of some of the fibres that turn upward in the column. Some seem to go to ganglion-cells of the grey matter or to the fibrillary plexus, others to the anterior nerve-roots. Through this commissure must also pass the fibres of the anterior pyramidal tract, and from it may come certain fibres that have been traced across the intermediate grey substance to the lateral column, where they assume a vertical direction in the pyramidal tract, so that part of the anterior commissure is to be regarded as a continuation

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descending antero-lateral tract referred to on p. 215—the relations of which are not yet quite distinct (see Biedl, 'Neur. Cent.,' 1895; Mott, 'Brain,' 1895; Risien Russell, 'Brain,' 1898).

[*] But Rossolimo ('Neur. Cent.,' 1898, p. 935) traces Gowers's antero-lateral tract in a case of tumour of the cord into the posterior corpora quadrigemina, the substantia nigra, and the globus pallidus. Of its different destination in monkeys there seems to be no doubt.

of the decussation of the pyramids. Lastly, a few fibres of the anterior commissure have been shown to come from the posterior nerve-roots.

The *grey commissure* contains very fine medullated nerve-fibres, a large number of which pass backwards in the posterior horn of each side; some appear to be continuous with the fibres of the posterior roots, while others probably end in the nerve-cells of the posterior horn. It is highly probable from clinical evidence that a considerable number of the posterior root-fibres (or fibres from cells in which the root-fibres end) cross to the other side, but no reliable experimental evidence of such a crossing has been published. Within the grey commissure is the *central canal* of the spinal cord, lined with epithelium, and surrounded by granular material, said to be similar in nature to that which caps the posterior horn; the two are, indeed, as we have seen (p. 206), continuous in the embryo. The canal is often filled up by epithelial débris in adult cords,* or, sometimes, is much larger than normal. The latter state probably depends on an arrest of development, for the layer of neuroglial tissue around it is much larger than usual.†

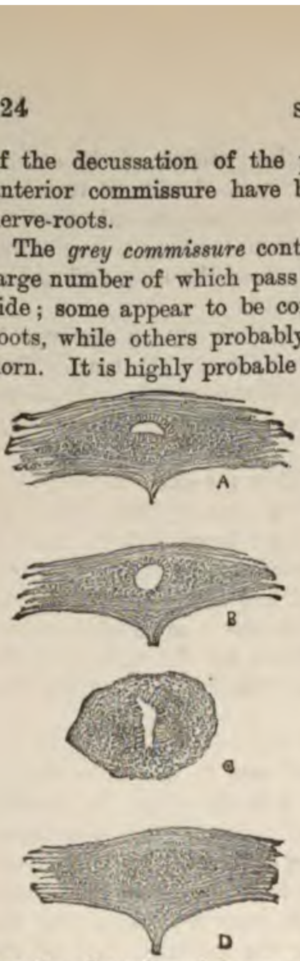

FIG. 82.—Central canal in normal cords. It has the form of a transverse slit in A, a vertical slit in C (from the conus medullaris), while in B it is circular. In each it is lined by columnar epithelium and surrounded by cellular elements, rounded, or angular from compression, mingled with granules. In D, which is from the same spinal cord as C, the position of the canal is occupied by a mass of nuclear tissue.

Root-fibres.—The course of the root-fibres has been already incidentally alluded to, but remains to be considered in a more systematic manner. The *anterior roots* pass through the anterior columns in bundles which are distributed through an area of the column approximately corresponding to the width of the anterior surface of the cornu. In the grey substance, the coarse fibres that constitute the chief part of each of these bundles pass in three different directions, outwards, inwards, and directly backwards in the middle of the horn, and thus cross each other and the fibres from the anterior commissure. Many of the fibres enter the groups of nerve-cells, and certainly end in these cells—or rather arise from them, each axis-cylinder being the prolonged chief process of the cell. Such cells often lie among the bundles outside the limit of the grey matter, and their processes can easily be traced forwards to the nerve-root and backwards into the grey matter. Some processes that pass outwards enter the lateral white column ("mixed zone"), and probably, after a short course in this, re-enter the grey matter at a higher or lower level. Some of the fibres that pass inwards may go through the commissure to the opposite anterior horn. These coarse fibres are probably all motor. Besides them, the nerve-roots contain fine fibres for the sympathetic which do not come from the cornual cells.

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* This obliteration of the canal has no pathological significance, but has been repeatedly described as one of the morbid changes in disease.

† Ciaglinski ('Neur. Cent.,' 1896) describes a long, diamond-shaped tract situated in the grey commissure between the ventral extremity of the posterior columns and the central canal, diminishing in volume as it ascends, but traceable to the cervical region. He believes that its fibres are derived from the posterior roots, and subserve the conduction of thermal and painful impressions.

The *posterior root-fibres* have been the subject of much investigation on account of their complicated arrangement and the importance that attaches to them from their degeneration in locomotor ataxy. Although arranged in a continuous vertical series at their surface attachment, the fibres are divisible into three sets, one of which passes through the *caput cornu posterioris* (Fig. 80), a second into the adjacent part of the postero-external column (Fig. 65), while a third set consists of fine fibres which at once change their direction, turning upwards, and keep together so as to form a zone (zone of Lissauer), which lies outside the extremity of the posterior horn in the cervical region, but lower down the cord between the caput and the surface of the cord.

The fibres of these three sets have only two immediate destinations—to the postero-median column and the posterior grey cornu. (1) Many of the fibres that enter the postero-external column (*a*) pass obliquely upwards and inwards to the postero-median column. This course is through the hinder part of the postero-external column, and in the lumbar region, where the roots enter at the bottom of their furrow, many fibres actually pass backwards into the adjacent angle of the column, and may be there seen to turn upward in their ascending course to the median column. It is this arrangement that leads to the peculiar wedge-like shape of the ascending degeneration just above a lesion (Figs. 77 and 78); (*b*) other fibres entering the postero-external column curve forwards and enter the posterior cornu, chiefly in front of its head. Some of these may end by interlacing with the processes of cells in Clarke's column or the anterior horns. The curves they describe are largest in the lumbar region, where they almost reach the postero-median column. (2) The fibres that enter the caput directly, pass through this to the posterior horn, some on their level of entrance (and these, in the lumbar region, have a curved course like the meridian lines on a globe), while others turn upward in compact bundles to pass forward into the cornu at a higher level. (3) The fibres of Lissauer's group also enter the horn, either through or in front of the caput, after a short ascending course.

Thus, except the fibres to the postero-median column (which ascend in it without decussating), all the other posterior root-fibres enter the posterior horn, chiefly on its inner side or through the caput, a few (Lissauer's) on its outer side.*

In the posterior horn, fibres of each set pass to the posterior commissure; others go to the posterior vesicular column (where this exists), while some pass forwards into the anterior cornu, chiefly towards the outer group of nerve-cells; a few pass towards the anterior commissure, and a considerable number probably end in the nerve-cells of the horn. The fibres that pass to the posterior vesicular column break up into branches which interlace with the dendrites of its cells. Many of those that pass towards the posterior commissure are probably also interrupted by nerve-cells in the posterior cornu. Thus the chief destination of the fibres that do not enter the postero-median column must be regarded as the anterior cornu of the same side and the opposite half of the cord; and this is probably true also of the numerous fibres that end in the nerve-cells of the cornu. The fibres from the vesicular tract to the direct cerebellar tract perhaps continue the path of the posterior root-fibres.

BLOOD-SUPPLY TO THE SPINAL CORD.—The arrangement of the arteries in the cord is not a matter, at present, of much practical importance. Nevertheless it is one on which some detailed facts are necessary, because it is a subject to which it is most desirable close attention should be given on account of the probability that vascular lesions are more frequent than our present knowledge indicates, and the relation of lesions to arterial regions should be carefully noted. The chief general fact is that the blood-supply is in part central, in part peripheral; the central supply is derived from the branches in the anterior median fissure, and embraces the grey matter, except the posterior horn; the peripheral supplies the posterior horn (except the neck) and the white substance.

The arterial blood is brought to the cord by small branches, derived from the vertebrals, intercostals, and other arteries, which reach the cord by the anterior and posterior roots. The anterior pass for the most part inwards to the anterior median fissure, where they are connected by vertical branches, continuous in direction, so as to form an *anterior spinal artery*. From this a series of branches pass backwards in the anterior median fissure, which may be called *anterior median arteries*, and are of great importance, supplying most of the grey matter. At the bottom of the fissure each divides into two branches, a right and left *commissural artery*, which passes outwards and backwards through the commissure, displacing its fibres (and hence, in section, the commissure often appears to be interrupted where the fibres are divided obliquely). At the end of the commissure each divides into an *anterior central artery*, which supplies most of the anterior horn, and a *posterior central*, which supplies the intermediate grey matter and the neck of the posterior horn, including the region of the posterior vesicular column. Each commissural artery, moreover, before dividing, gives off a branch which immediately bifurcates into an upward and downward vessel, each continuous with a corresponding branch from the next commissural artery above and below—the *anastomotic artery*. This effects a vertical continuity of anastomoses within the cord, like that of the anterior spinal artery outside the cord.

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* The fibres that pass to the postero-external column have been termed the "median group," and the rest of the root-fibres the "lateral group," those entering the caput being distinguished as an "intermediate group." But each investigator describes much the same facts in a different method, and adopts an original nomenclature, which renders it better at present to keep the facts more prominent in the mind than the names.

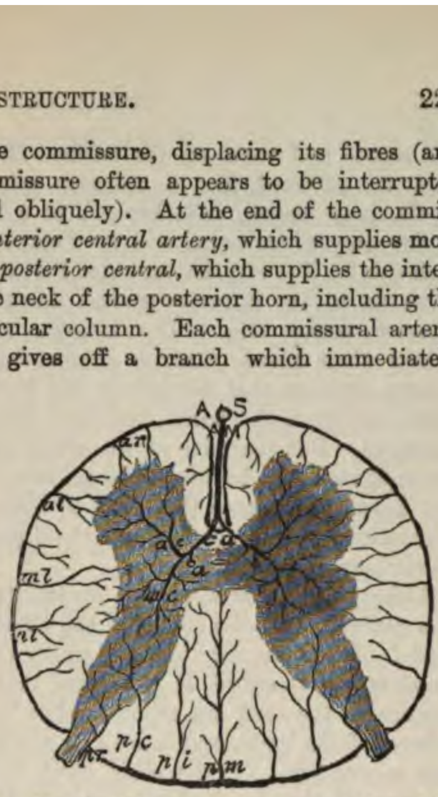

FIG. 83.—Semi-diagrammatic representation of the arteries of the spinal cord; A S, anterior spinal. *Central arteries.*—A M, anterior median; ***c a***, between the right and left commissural arteries; ***a***, anastomotic artery, divided transversely, to which a branch goes from the commissural artery, which then divides into ***a c***, anterior cornual, and ***m c***, mid-cornual arteries. *Peripheral arteries.*—***p m***, posterior median; ***p i***, post-intermediate; ***p c***, posterior cornual; ***p r***, posterior radicular; ***p l***, ***m l***, ***a l***, regions of the posterior, middle, and anterior lateral branches; ***a r***, anterior radicular. In the right half of the figure the more deeply shaded part indicates the region supplied by the central arteries.

The *peripheral arteries* pass inwards from the surface. A *posterior median* artery courses in the median septum, giving branches to each side, and an *intermediate septal artery* passes in the corresponding septum outside the post-median column. Midway between this and the root-furrow a branch enters and passes through the postero-external column to the posterior horn, which it enters in front of the caput and chiefly supplies—the *posterior cornual artery;* while a *posterior radicular artery* passes in on the inner side of the posterior root, and is distributed to this and the caput. A series of *anterior radicular* arteries enter with the anterior roots, and between them and the posterior horn are *anterior*, *middle*, and *posterior lateral* arteries in corresponding parts of the lateral column. The branches into the lateral column do not reach the grey substance; the branches in the grey substance extend into the inner part of the lateral column.

The *veins* of the cord correspond in the main to the arteries, but a large proportion of the blood passes into the peripheral system. Some, especially those of the white substance, pass outwards along the septa; and those on the surface of the sides and back of the cord are gathered into a *posterior spinal vein*. Others, especially of the grey matter, pass inwards to a large vertical vein that lies in the grey commissure, a little distance from the central canal on each side (*v*. Fig. 80; compare also Fig. 65), from which branches pass forwards to the anterior median fissure, and through this to an *anterior spinal vein*. The anterior and posterior spinal veins deliver their blood, through communicating veins, into the large venous plexuses that lie outside the dura mater, and which receive blood also from the bones, and from the structures and skin behind the spine. But the veins of the spinal cord cannot be injected from these plexuses, not because there are valves in the connecting branches, but apparently because they form so trifling a proportion of the total connections. From the plexuses blood passes to the various vertebral, cervical, and intercostal veins.

Although there is a vertical continuity of the vessels of the cord, it is probable that the course of the circulation is, in the main, horizontal. From the very tortuous course of the path by which arterial blood enters the cord, it is evident that the pressure to which the arteries of the cord are exposed must be far lower than that in the arteries of the brain, and hence they are far less liable to degeneration and to rupture. On the other hand, the conditions that prevent an injection of the plexus of veins outside the dura mater from passing into the veins of the cord, must save the latter from the extreme over-distension to which they would otherwise be liable when there is a hindrance to the return of blood from the plexus.

FUNCTIONS OF THE SPINAL CORD.

We are now in a position to consider the functions of the spinal cord, and the structures by which they are subserved. The functions are these: (1) The spinal cord conducts motor nerve impulses from the brain and sensory impulses to it. (2) It constitutes a series of centres for reflex action. (3) It contains certain centres that ultimately govern nervous action in structures under the immediate control of the sympathetic system, the bladder, rectum, blood-vessels, &c. (4) It influences the nutrition in all parts to which its nerves extend.

Motor Conduction.—The chief facts regarding the physiology of the motor path have been necessarily considered in the account of its anatomy. We have already seen that the path of motor impulses is through the pyramidal fibres (in the anterior and lateral pyramidal tracts), the anterior cornua, and the anterior nerve-roots. The ganglion-cells form part of the path, and so also probably does that part of the plexus of fibrillæ in the cornu which intervenes between the pyramidal fibres and the ganglion-cells. We have seen that the crossed relation of the cerebral hemisphere with the limbs is subserved by the decussation at the pyramids, and apparently to a slight extent above the chief crossing, since some degenerated fibres have been seen in the anterior pyramid opposite to the lesion of the brain. We have seen also that we are still ignorant of the mode of connection of the motor cells with the cerebral hemisphere of the same side in the case of the muscles that habitually act with their fellows on the opposite. These can be acted on by either hemisphere in degree that approaches equality in proportion as they act together, and is almost complete in muscles such as the intercostals, which can only act together. It is great in the case of all the trunk muscles and of those of the leg, less in those of the arm. This relation is further considered in the account of hemiplegia in Vol. II, to which it specially pertains. The variations in the decussation of the chief set of fibres suggest that the variations in observed facts bearing on the question may merely represent variations that exist in nature. The connection may be subserved by the fibres already mentioned that never decussate, but pass into the lateral pyramidal tract of the same side.

The large fibres of the anterior motor nerve-roots are far more numerous than the fibres of the pyramidal tracts, and so also are the cells of the anterior horn, from which the large root-fibres proceed. Nevertheless it is probable that all these cells and root-fibres can be excited through these tracts. Hence many cells must be related to each of the pyramidal fibres. A complex mechanism is no doubt established by the interlacement of the terminal fibrillæ of the pyramidal axons with the dendrites of the anterior cornual cells. This mechanism is such that the stimulation of certain pyramidal fibres excites to action a much greater number of nerve-cells, so connected and arranged as to produce, through the related nerve-roots, a complex movement, in which many muscles may take part. The nerve-cells thus connected may not all be at precisely the same level, and only some of those at a given level may be thus associated. The simpler the movements, and the fewer their possible variations, the fewer pyramidal fibres may suffice for the production of the movements. Thus it is conceivable that for such movements as those of the intercostal muscles, which are simple and constant, very few pyramidal fibres may suffice, whereas a much larger number must be necessary for the highly variable movements of the hand. It is probable also that the pyramidal impulses influence other neurons than those directly subserving muscular contraction. By means of such intermediate or associational neurons these impulses are enabled to effect a more complex and varying movement than could otherwise be produced.

REPRESENTATION OF MOVEMENTS IN THE SPINAL CORD.—It important to inquire how far we can identify these various motor mechanisms of the anterior grey matter. There are several sources of information on this subject. We have already seen that the arrangement of fibres in the anterior roots is such as to associate certain movements with certain nerve-roots, and it is a reasonable assumption that this corresponds, to some extent at least, with the arrangement in the grey matter. The sources of our information on this point are the limited facts of anatomy, rare cases of restricted disease of the nerve-roots in man, and especially the experiments of Ferrier and Yeo on monkeys. We have already considered the indications that these experiments afford, and their suggestiveness, and we have seen that their value is relative rather than absolute. As regards the grey matter, we must remember also that there may not be a strict horizontal correspondence between the nerve-roots and the nerve-cells, because it is probable that some root-fibres enter the antero-lateral white columns, and are connected with nerve-cells at a higher or lower level than that at which they leave the cord. Another source of information is the degeneration of nerve-cells that follows slowly on an amputation of a limb, and the condition of the spinal cord in congenital absence of part of a limb. The last and most important source of information is supplied by cases of limited disease of the anterior cornua, in which the position of the lesion and the distribution of the resulting palsy can be compared. Destruction of nerve-cells causes degeneration of the motor fibres proceeding from them, and wasting of the muscles to which those motor fibres proceed. This alone affords us certain knowledge. Cases of clear significance are, however, rare, and it will be long before our knowledge can be complete. Meanwhile we may learn something of the central association of muscles by observing what muscles are most frequently paralysed together by such disease. This subject has been carefully studied by E. Remak* and by Thorburn,† and many valuable isolated observations have been published. It is only the associations which are frequent that can be allowed significance, because it is not uncommon to have more than one focus of disease in the grey matter. The following summary presents the conclusions that seem probable from the evidence at present available.‡

The various facts for the most part fully confirm the conclusion of

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* 'Archiv f. Psychiatrie,' vol. ix.

† 'Brain,' January 7th, 1887, and October, 1888. The facts were observed on cases of injury. He associates each muscle with a single spinal nerve, but these nerves are only collections of roots that are severally continuous, as are the groups of nerve-cells within the cord, and it is certain that the roots related to many of the muscles go into adjacent spinal nerves.

‡ A useful summary of the evidence up to the middle of 1884 was given by Dr. Allen Starr, in the 'American Journal of Neurology,' Aug. and Nov., 1884. The subject is a tempting one for theory, but it is very important to keep to the solid ground of facts, however limited it may be in extent.

Remak, corroborated by the experiments of Ferrier and Yeo, that most movements and muscles are represented in vertical tracts, and the whole anterior grey matter, at any one nerve-segment, contains cells that are concerned with different movements. An extensive lesion of small vertical extent may thus weaken many movements, but abolish none. The special representation of the muscular functions, *i. e.* of definite movements, is no doubt related to special groups of nerve-cells, but a single group may be concerned in more than one associated movement. Different groups are probably intimately connected, because we know that the contraction of any muscle is accompanied by a slighter but proportioned contraction of its antagonists. Anatomical connections in both spinal cord and brain doubtless underlie this association.

Cervical Region.—The highest cervical region apparently contains centres for the small rotators of the head, and the depressors of the hyoid bone were found by Beevor and Horsley to be related to the first two pairs of nerves, the sterno-hyoid and sterno-thyroid chiefly to the first and the omohyoid to the second.* The upper part also contains centres for the muscles of the neck, especially for the sterno-mastoid and upper part of the trapezius. The diaphragm is probably represented in the grey matter at the level of the roots of the phrenic nerve, the fourth cervical. In the brachial region of the cervical enlargement, the muscles of the shoulder are represented chiefly in the upper part; the intrinsic muscles of the hand in the lower part. The flexors of the elbow are represented above its extensors, and the supinators and extensors of the wrist above the flexors of the wrist.

Deltoid, scapular muscles, pectoralis, and ***serratus.***—The fifth and sixth segments, for the most part the sixth, and probably the outer group of nerve-cells.† The centres for the two parts of the pectoralis are separate, and are associated—the clavicular with that of the serratus magnus, the costal with that of the latissimus dorsi (see p. 37). These associated muscles are represented near together, but not at the same spot; the association is often reproduced in disease of the anterior cornu, the clavicular part being affected together with the serratus, but one muscle may suffer without that which is commonly associated with it.

The ***Flexors of the elbow*** and ***supinators*** probably correspond nearly in level with the deltoid. The whole of this series of muscles may be affected alone by disease of the nerve-roots (see p. 38) or of the grey matter, and then we have the "upper arm type" of palsy, described first by Erb.

Extensor of the elbow (triceps) : probably the middle of the brachial region, chiefly the seventh segment, extending down to the eighth, and perhaps up to the sixth, possibly chiefly in the outer group of nerve-cells.

The ***Extensors of the wrist*** are represented above those of the elbow; probably chiefly at the sixth or sixth and seventh segments, and not from the

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* Chiefly the posterior belly of the muscle, by a branch joining the glosso-pharyngeal. The other muscles named (including slightly the anterior belly of the omohyoid) are innervated from the first cervical by a branch joining the hypoglossal.

† Thorburn (loc. cit.) refers the infra- and supra-spinati with much probability to the fourth pair of nerves.

postero-external, but from the other groups. *Flexors of the wrist:* below the extensors, nearly on the same level as the extensors of the elbow, at the seventh and eighth segments, and probably also from one of the outer groups of cells. The *pronators* have nearly the same representation as the flexors of the wrist.

Long Extensors of the fingers: upper part of the brachial region, about the level of the sixth or between it and the seventh segment; probably from one of the anterior groups (not from the postero-lateral group). *Long flexors of the fingers:* below the extensors; probably seventh or eighth segment.

Intrinsic muscles of the hand: lowest part of the brachial region, and the thenar muscles a little higher than the interossei; the latter being represented in the first dorsal segment.* The intrinsic muscles (like the long extensors) are probably related less to the outer than to the inner and anterior groups. The two sets of muscles are related both in action and in central connection, as was illustrated by a case of concussion-lesion under my care, in which the two outer interossei were paralysed and wasted, and also the part of the long extensor supplying the two outer fingers, but no other muscles.

Lumbar Enlargement.—Our knowledge of the representation of muscles in the lumbar enlargement is very slight, and we have scarcely any definite facts concerning their relation to the cell-groups. The postero-external group preponderates over the others in size, even more than in the cervical cord, and doubtless also in importance. The few pathological observations of changes in the several groups are not altogether consistent. The chief facts available relate to the probable segmental level and association of the centres, and are as follows:

Cremaster, second lumbar segment. *Psoas*, second, *iliacus*, third lumbar; the two muscles (one in function) are probably related to a group of cells extending through both segments. *Adductors*, fourth lumbar segment. *Gluteal muscles* (extensors of hip), fourth and fifth lumbar segments.

Extensors of knee, third and fourth lumbar segments, probably from the same group as the flexors of the hip; the two sets of muscles are often affected together, and sometimes the abductors suffer with them. The *Sartorius* is probably related to the third segment, but not to the same cell-group as the extensors of the knee (to which, indeed, it does not belong; see p. 47). It usually escapes in atrophic palsy of the extensors. *Flexors of knee*, fifth lumbar and first sacral segments. Their centre is thus below and certainly distinct from that of the extensors.

The muscles of the lower leg, moving the foot and toes, are related to the fourth and fifth lumbar and the first sacral segments. The *calf muscles*, the *tibialis anticus*, and the *peroneus longus* have certainly separate centres, and are often affected separately. The tibialis anticus may suffer alone, or may alone escape when the other muscles in front of the leg are paralysed. The muscles of the calf may be affected when those in front of the leg are not. It is probable that the peroneus longus is the highest in central representation, and is related to the fourth lumbar segment.

The *intrinsic muscles of the foot*, especially the interossei, are the lowest in

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* This is the indication of the majority of cases. Nevertheless Sahli has recorded a case of atrophy of all the intrinsic muscles of the hand, in which the lesion did not extend below the seventh segment; the eighth and first dorsal were perfectly normal. I have seen a case of atrophy limited to the deltoid and intrinsic muscles of the hand, due to an acute process, probably hæmorrhage, but in this case there may have been two lesions.

central relations, being connected with the second sacral segment. The position of their centres, in the lowest part of the lumbar enlargement, thus corresponds to that of the analogous muscles of the hand, in the lowest part of the cervical enlargement.

SENSORY CONDUCTION.—Our knowledge of the sensory path in the cord is far less definite than that of the motor path, in spite of the fact that it has been the subject of a large number of experimental investigations. The subject is a very difficult one for experimental solution, on account of the difficulty of ascertaining the condition of sensation in animals. The indications afforded by disease are equivocal for another reason. We have seen (p. 78) that, in the sensory nerves, a lesion which permanently interrupts motor conduction may scarcely affect sensory conduction, and the same thing is apparently true of the spinal cord. If loss of feeling results from an acute lesion, it often quickly passes away, although motor palsy remains complete and absolute, and this when all the elements of the cord seem equally implicated in the morbid process. In cases of such chronic disease as compression, which when slow must act on all the structures in nearly equal degree, the same escape of sensory conduction is often observed. Difficulties are encountered whatever mode of interpretation of the phenomenon we adopt, and the fact of importance to our present problem is that we are not yet able strictly to compare structural and functional damage when the former is moderate. When the damage has been slowly produced it may be apparently great, and yet function may not be much impaired, especially the lower function of conduction of nerve-force. The significance of these facts as regards the problem of sensory conduction in the cord is that we must be cautious in drawing any conclusion from the persistence of sensation. We cannot infer, because supposed sensory tracts are visibly diseased in a case in which there was no loss of sensibility, that these tracts have not the function assigned to them, unless we can feel sure that all the axis-cylinders are destroyed. This we can seldom do; and for this reason, and because morbid processes are often wide in distribution, the evidence that can be obtained by comparing the position of lesions with the sensory symptoms they produce is limited and uncertain. Other sources of information are the effect of partial lesions of the cord, intense in degree, but limited in area, especially traumatic lesions affecting one half of the cord or part of one half. Cases of this kind are of high value, but in few, at present, have the extent of lesion and the range of symptoms been accurately ascertained.

Hence physiologists have relied chiefly on the results of experiments on animals. Unfortunately the help these can give is also reduced to a literal minimum by the difficulties of experimentation and of interpretation—the physical difficulties on the one hand, the functional perplexities on the other. Some clear facts have been ascertained, and other results have been obtained that still wait certain interpretation. It seems certain, in the first place, that sensation in any particular region of the skin is associated with the functional activity of a definite portion of the opposite cerebral cortex. Anatomical evidence shows that the axons belonging to different centripetal tracts cross the middle line at various levels in the cord, medulla, and mid-brain. The early experiments of Brown-Séquard led him to conclude that the chief part of the sensory path decussates soon after entering the cord, and with this view pathological facts in general agree. The consensus of evidence, however, points to a less sudden and uniform crossing than was formerly assumed. The axons entering from the periphery do not as a rule pass directly across the cord, but break up in the grey matter, whence, possibly after one or more relays, the conducting path is continued on the opposite side. It will be seen that these considerations suggest that sensory conduction is a function of the lateral and antero-lateral tracts rather than of the posterior columns, which run uncrossed to the region of the medulla. This is quite in accordance with what is found both clinically and experimentally. For the posterior median fibres may be extensively degenerated when sensation is unimpaired, a fact of which we shall see the probable meaning presently; and, on the other hand, Osawa has shown that section across the whole cord, with the exception of the posterior column, completely abolishes sensation. That this abolition is incomplete unless the grey matter is completely divided was found also by Schiff; the important part played by the grey matter in sensory conduction is thus confirmed.

The subject, however, at the present time is too far from even the semblance of settlement to make a discussion of the evidence worth the space it would need, especially in a work dealing with practical medicine.* It is only desirable, therefore, to point out certain leading facts and leading considerations.

Clinical evidence, largely based upon cases of syringomyelia, points to the grey matter as the seat of conduction of painful impressions. Experiments suggest that impressions which act upon the vaso-motor centrè pass up in the lateral columns—in animals chiefly on the side of the cord opposite to that on which they are produced, slightly on the same side. Sensations of touch appear to be conducted mainly in the lateral columns and grey matter. In man the paths for both touch and pain are on the opposite side of the cord.

We may accept as certain the fact that the tactile or painful impulse we feel crosses the middle line soon after entering the cord; it is probable that some impulses that we do not feel do not cross. We must never forget that there is strong reason to believe that only a minority of the impulses that traverse afferent nerves affect our consciousness.

The participation of the grey matter in the conduction of afferent impressions has been fully confirmed by the facts as to the course of centripetal fibres in the cord revealed by the recent improvements in histological technique. That sensation escapes in anterior polio-myelitis points to the posterior part of the grey matter as the seat of this conduction, and this again is in agreement with both histological and experimental† evidence.

The assumption that a certain number of centripetal axons exist in the central grey matter is of great service in studying the problems of sensory conduction. Still, they are not numerous enough to save us from the difficulty that the fibres we can look to are, taken altogether, insufficient in number to subserve the marvellous differentiation of sensation in the periphery—differentiation in quality of feeling and especially in locality of feeling. But a difficulty may become inoperative, without ceasing to exist, by being absorbed in a still larger difficulty of the same kind. If we avail ourselves of all the structures of the spinal cord that can conceivably subserve upward conduction, we fail to find room for the separate conduction of impulses which, nevertheless, the spinal cord must separately convey.

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* Thus, for example, two of the latest series of experiments upon monkeys, those of Mott and Turner, yield conflicting results. The latter are mainly confirmatory of those of Brown-Séquard; while, according to the former, touch and the muscular sense are mainly transmitted up along the side of the cord at which they enter, but the paths of pain and temperature pass up on both sides.

† *E. g.* the researches of Schiff and Ciaglinski (see later).

On only one hypothesis is it possible to conceive such conduction. It has already been pointed out that the increased power of the microscope has shown us that each axis-cylinder, so called, is not a simple body, but a compound structure, composed of fibrillæ, and comparable, in point of fact, to a bundle of nerve-fibres. What was formerly regarded as division of an axis-cylinder is now known to be the separation of its ultimate fibrillæ, and this has been shown to occur in both motor and sensory axons, and, to a certain extent, along their course (collaterally) as well as at their terminations. It seems to follow of necessity that each sensory area of distinct perception must be subserved by one of these fibrillæ. It is conceivable that the impulses may keep each to its own fibrillary path, however many fibrillæ an axis-cylinder may contain. We have, indeed, seen that such fibrillæ keep separate even in traversing a nerve-cell. It is only by such an arrangement that it seems possible for the sensory impressions from skin and tissue, of touch, pain, and temperature, to be carried to the brain so as separately to excite certain nerve-cells of the cortex.

If these considerations have weight we cannot exclude the lateral and posterior columns from the function of sensory conduction on account of the scanty fibres we can discern to subserve the function, and may consider what evidence on the point disease and injury afford in man.

Unilateral lesions of the spinal cord, which cause loss of sensibility of the skin, do so on the side opposite to the lesion. This is true of all forms of sensibility. They thus prove, beyond doubt, the decussation in the spinal cord of the path from the cutaneous nerves of pain, of touch, and of temperature. An analysis of the cases in which both effect and lesion have been exactly ascertained—which will be considered more fully when these lesions are described—suggests certain other conclusions regarding the position of these paths. The facts suggest, first, that the paths for sensations of pain and of touch are not near together. Sensibility to pain has been lost in almost all recorded cases, but that to touch in only two thirds. They suggest, secondly, that the two paths for tactile sensibility are nearer together than are the two paths for pain. In no case of chiefly unilateral lesion has sensibility to pain been lost on both sides, whereas in two recorded cases sensibility to pain was lost on the side opposite to the lesion, while that to touch was lost on both sides.*

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* It is possible that tactile sensibility may have been lost, in other cases, on the second side, *i. e.* on the side of the lesion, and the loss may have escaped notice in consequence of the exaltation of sensibility which is commonly present, and which is presumably due to the nerves of common sensibility.

These conclusions, moreover, are supported by the facts of two important cases of injury to the cord which afford direct localising indications. One is a case, recorded by myself,* in which the spinal cord in the upper cervical region was damaged by a small spiculum of bone being driven against it by a bullet, which, entering the mouth, lodged in the body of the third cervical vertebra. The extent of the lesion of the cord is shown in Fig. 84. The chief injury is clearly to the lateral column and grey matter, the posterior column being merely swollen, apparently by œdema. The affected part was the seat of hæmorrhage and hæmorrhagic infiltration. The effect was entire loss of sensibility to pain on the opposite side without any impairment of tactile sensibility. To this the other case, recorded by Müller,† is almost a complement. A stab-wound divided the whole of one half of the cord, including the posterior column, and also the posterior column of the other side almost up to the nerve-roots; the grey matter of this side escaped. The whole of both postero-median columns was thus divided. The effect was loss of sensibility to pain on the side opposite to the lesion, and of that to touch on both sides.

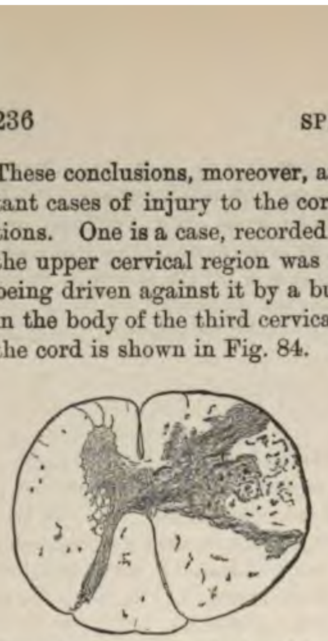

FIG. 84.—Section of spinal cord between the second and third cervical nerves, contused by gunshot injury.

Thus there is strong reason to believe that the path for sensibility to pain does not pass in the posterior columns. The evidence of disease (syringomyelia) points to the grey matter as at any rate the main seat of this path. We have already seen that the escape of sensation in anterior polio-myelitis indicates that it must pass along the posterior part of the grey matter, and this accords well with experimental evidence. Thus Schiff found that section through the whole cord except the posterior columns abolished sensation to pain below the lesion, whereas if a trace of the posterior part of the grey matter was left this sensation was still transmitted, though delayed. Ciaglinski‡ describes an ascending tract, found experimentally in the dog and verified histologically in man, between the postero-median column and the central canal, and regards this as the path for impressions of pain and temperature. As to the path of tactile sensibility we must wait for further evidence. The fibres that degenerate upwards in the median columns, passing to them from the external posterior columns, and constituting so large a proportion of their mass, apparently come from the muscles. We may, however, remember that many fibres pass to the posterior columns from the grey matter, and also from the posterior commissure in the middle line; some of the former may also come from the posterior commissure, and both these sets of fibres may have crossed in the middle line. It is possible that these fibres conduct tactile sensibility, and that they are mingled with those that conduct muscular sensibility. This theory, however, can only be regarded as a suggestion for future observations; it must be confessed that the results of experiment, which are, however, for reasons already stated, not so valid as clinical observations, tend to indicate the implication of the lateral and antero-lateral columns in the conduction of tactile sensations.

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* 'Clinical Society's Trans.,' vol. xi, 1878, p. 24.

† 'Beiträge zur path. Anat. und Phys. der Ruckenm.,' Leipzig, 1871. Abstracts, which supplement each other, and, together, give a full account of the case, will be found in Köbner's article on unilateral lesions, 'Deut. Arch. f. klin. Med.,' 1877, Bd. xix, p. 190; and in 'Virchow's Jahresbericht,' 1871, Bd. i, p. 152.

‡ Ciaglinski, 'Neurol. Centralbl.,' 1896.

The conduction of impulses from the muscles is certainly in the posterior median columns. This is clearly shown by the facts of the pathology of tabes. The function of the direct cerebellar tract has still to be demonstrated. There are certain resemblances between its fibre-relations and those of the posterior median columns which give countenance to Flechsig's theory that it conveys impressions from the muscles of the lower part of the trunk and between the trunk and lower limbs. But the origin of its fibres from the cells of the posterior vesicular column, and the mystery attaching to the latter, give additional obscurity to this structure. The fine nerve-plexus between the cells seems connected with fibres of the posterior roots, and this plexus is said to atrophy early in locomotor ataxy.[*] The obscurity will, indeed, be dispelled if we can accept the speculation of Sherrington that this cylinder is really part of the series of ganglia on the posterior nerve-roots, which occupies a position within the spinal cord, and is continuous instead of broken into ganglia.

The path for sensations of temperature is still unknown. The fact that this sense is frequently impaired with sensibility to pain suggests that the two paths may be near together in the grey matter. More than this cannot be said.

But we have to consider another problem in our study of the sensory path. The same impression that is felt may excite a reflex action. For this, afferent root-fibres must end in the grey matter of the cord. Are the two functions subserved by the same or by different fibres? A similar question presents itself with regard to the muscle nerves. If the ascending degeneration in the postero-median column, when the cauda equina is diseased, is admitted, as it must be, as proof that these root-fibres pass up without interruption, the muscle-reflex action must be subserved by other fibres. For it, however, comparatively few may suffice. With regard to the other sensory fibres, the evidence at present suggests that all undergo interruption in the spinal cord, since no other paths have yet been found degenerated when a lesion has been confined to the nerve-roots. If so, the same fibres probably subserve sensation and reflex action, the dendrites of the nerve-cells providing a mechanism for the division of the path and diversion of the impulses. No impression, in health, causes a true reflex action that does not also produce a conscious sensation.

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[*] Lissauer, Oppenheim, &c. See Mott, "Bipolar Cells of the Spinal Cord," 'Brain,' part lii, 1890.

Our knowledge of the serial representation of cutaneous sensibility in the spinal cord has of late been much increased by the careful researches of Head, Thorburn, Starr, Dana, and others. It is probable that there is a progressive representation of the skin in the posterior nerve-cells without relation to the nerve-trunks, and with only a slight relation to the nerve-roots. Correspondence with the latter must not be assumed. In the roots the serial representation is along the axis of the limb, not across it, as it probably is in the nerve-cells. In the dorsal cord, ascending disease often causes an affection of sensation which extends around the trunk at higher and higher levels. In the limbs we have indications of the same arrangement: the soles and palms may be affected alone, and anæsthesia may extend on the limb to a certain level, irrespective of nerve distribution. This is often seen in the legs. I have known anæsthesia in the arms, due to disease of the cord, to cease midway between shoulder and elbow, at the same level around each arm. Disease of the cord may affect sensation on the tips of the fingers only. It is probable that the ascending course of many of the root-fibres subserves a rearrangement of representation in the cells, and provides also for a special connection of the tactile nerves from each part of the skin with the motor cells for the muscles beneath—so conspicuous in many of the cutaneous reflexes.

Reflex Action.—The grey matter of the spinal cord constitutes a series of reflex centres, some of which must be of considerable vertical extent and much complexity. They are subserved not only by the grey matter, but by the short fibres that in the several white columns connect the grey matter at different levels. The passage of the root-fibres upwards and downwards in the cord, before they turn into the grey matter (conspicuous in the posterior roots), also, as we have noted, constitutes an arrangement for spreading the afferent impulses through a considerable vertical extent of the cord.

We have already seen (p. 19) that we must distinguish two forms of reflex action—the cutaneous reflex action and the muscle-reflex action, the latter producing the "myotatic irritability" which, as we assume, determines the so-called "tendon-reflex contractions." The first form of reflex action is not, however, limited to impressions on the cutaneous nerves. It may be produced by stimulation of the nerves supplying the deeper structures, including those of the tendons and muscles. The attempt to obtain the knee-jerk may cause a true reflex action as well as its special effect, the two being separated by an appreciable interval of time (see Locomotor Ataxy). Reflex action from the skin is apparently subserved by all the sensory nerves, since it may continue, although the nerves for either pain or touch have ceased to act, in consequence of those degenerative processes by which function is sometimes abolished in as complete degree and with as precise limitation as the physiological inquirer could desire.

The central process concerned in reflex action must take place between the posterior nerve-roots, by which the sensory impulses reach the cord, and the anterior roots, by which the motor impulses leave it. The motor ganglion-cells form part of the reflex centre, and the rest of the centre is doubtless constituted by the fibrillary plexus formed by the inosculation of their dendrites and those of the sensory nerve-cells. The whole path between and including the in- and out-bearing nerves may be spoken of as the "reflex arc;" the grey matter concerned being called the "reflex centre." In this "centre" the sensory impulse is transformed into—or rather gives rise to—a motor impulse, usually much greater in the degree of its energy. Within the centre there are paths established by the connections between the nerve structures, of which only a few are in functional use. It is easy to conceive that the functional action takes place more readily in some lines than in others, and these lines of easiest action are often spoken of as "lines of least resistance," and are said to determine the form of the reflex action. In its simplest form a "reflex arc" consists of two neurons, a centripetal and a centrifugal, between the dendrites of which the transformation of the impulse takes place. More powerful stimulation brings also into action the dendrites of the centripetal neuron which are associated with other neurons than the centrifugal already mentioned, and hence conduces to a wider area of motor discharge.

The stronger (within limits) the afferent stimulus, the more widely does the process spread in the centre, and the more extensive is the movement produced. Thus a slight touch on the sole may cause only a movement of the foot, while a prick may cause a movement of the whole limb. At the same time there is much more than degree involved in the difference between these two stimuli. The impression that is felt as pain causes a quick simple movement of the part, in which chiefly the flexors are involved. It is a movement obviously to withdraw from harm, and it is proportioned, in energy and extent, to the intensity of the stimulation. To this, however, there is one limitation:—a strong impression of pain may prevent all reflex movement by exerting an inhibitory influence on the centres. On the other hand, a tactile impression causes a much more complex movement, which does not bear a simple relation, in extent and energy, to the degree of the sensory impulse, but is also influenced by the quality of the impression at the place at which it is produced. Thus the reflex movement produced from the palm or sole is especially elaborate; it may present an indication of the central relations by which the purposed use of the extremities is facilitated. The effect of all painful impressions is similar, whether these are produced through the nerves of the skin or those of deeper structures. The reflex action caused by pinching a tendon is just the same as that caused by a pinch of the skin over the tendon. Even impressions from the afferent muscle-nerves cause a similar reflex movement. Thus there is no real distinction between the deep and superficial true reflex action.

The muscle-reflex action which is assumed to exist between the afferent and efferent nerves of a muscle (and to underlie the myotatic irritability which permits the knee-jerk, &c.) has been already considered in discussing the theory of this irritability. It is apparently a uniformly simple relation between the two sets of nerves for the same part of a muscle; the reflex action is confined to these, and does not spread through a wider extent of the grey matter, as that from the skin frequently does. When a muscle is made tense, or its tendon is tapped, the mechanical stimulation influences all the afferent nerves or all the muscular fibres, and the whole muscle necessarily responds. The motor ganglion-cells of the anterior horn must form part of the centre for each form of reflex action, but that portion of the centre which intervenes between the afferent nerves in the posterior root and the motor cells must be distinct in part or altogether. Beyond this we can say nothing of the relation of the two centres.

These reflex centres are subject to control by higher centres, as are all lower centres in the nervous system. All true reflex action, *e. g.* that from the skin excited by a touch or prick, is controlled and kept moderate by an influence from the brain, for it becomes excessive when the influence of the brain is stopped, as by a lesion interrupting the spinal cord; it is increased in all parts that are connected with the cord below the lesion. We have no precise knowledge of the seat of the controlling centre or of the path by which the control is exerted. In frogs, however, the restraint seems to be exerted by the optic lobes; in man it is less likely to be exerted by the homologous corpora quadrigemina than by the optic thalamus. The mechanism must, however, be complex, since the restraining influence may be increased by disease in some parts of the brain; many cerebral lesions which cause hemiplegia cause also a diminution of cutaneous reflex action on the paralysed side. The effect seems to indicate that the cerebral centre which controls reflex action is itself habitually controlled by a higher centre, perhaps in the cortex. When this is destroyed, or the path from it is interrupted, the controlling centre passes into a condition of increased activity, and the reflex action is lessened. This is a theory only, but it seems impossible otherwise to explain the facts.

The muscle-reflex centres seem also to be under the control of other centres, because, if there is disease of the cord higher up, they also pass into a condition of increased activity. But the phenomena of their disturbance present three important points of difference from those of common (*e. g.* cutaneous) reflex action. (1) We know with certainty the path disease of which determines their excess: it is the pyramidal tract. (2) Disease of the brain does not cause a permanent diminution in the action of the muscle-reflex centres, as it so frequently does in the case of the centres for cutaneous reflex action.* On the contrary, disease of the pyramidal fibres in the brain causes the same excess of this form of reflex action as does disease of the pyramidal fibres in the cord. (3) The excess does not, as a rule, quickly follow an interruption of the path from the brain, as does the excess of superficial reflex action. There may, indeed, be an immediate, usually temporary, excess, coinciding with "early rigidity" from the irritation of the pyramidal fibres, but the permanent excess comes on at the end of a week or ten days, and gradually increases.

The explanations may be these. The slowness with which the reflex action usually becomes excessive is conspicuous, and its significance must be considerable. The lesion of the pyramidal fibres causes their degeneration below the lesion, and this must involve their termination in the grey matter. The excess of action of the centres may be the result of the degeneration of the terminal structures, which may constitute a controlling mechanism that normally restrains the activity of the muscle-reflex centres. The interval which elapses before the excess is manifested may be due to the fact that there is little tendency to increased activity; that but slight control is habitually exercised or needed; and that the capacity for over-action is gradually developed and gradually increases—increasing capacity being developed by the increased activity.

It is probable that the voluntary impulses pass from the termination of the pyramidal fibres to the motor cells through structures of which this muscle-reflex centre forms part, and that therefore its elements must be in part excited to action by pyramidal impulses—a conclusion quite compatible with a restraining influence exerted by these fibres or their endings when no volitional impulses descend them.

The chief local forms of superficial and muscle-reflex action have been already described (see p. 19, *et seq.*). Their centres are found in the corresponding segments of the cord shown in the table at p. 252.

ADAPTATION TO POSTURE.—Every difference in posture involves a difference in the distance between the attachments of the muscles of the part. When the muscles are at rest they present always a certain state of "tone," which, being equal in every posture, must vary automatically with every change of posture, however brought about. In passive movements the muscles that are elongated and those that are shortened adapt themselves to the change as it proceeds. Apparently the increased tension on the former makes them yield to the movement, and the lessened tension on the latter makes them contract, each set thus preserving its proper tone. The tone of a muscle seems to be produced by a reflex process, due to a certain state of the motor cells regulated by the impressions from the afferent muscle-nerves. These, as we have seen, are produced by two mechanical influences, the lateral pressure of the fibres (as in the pain of cramp) and the tension on the muscle. An increase of the latter, in passive movement, seems to lessen the activity of the cells and permit the fibres to elongate; a diminution increases the activity of the cells and causes the fibres to shorten. It further appears that the tone of a muscle is influenced through the spinal cord by the condition of its antagonists. Thus the knee-jerk may be increased by cutting the sciatic nerve—*i.e.* the nerve to the flexors, or diminished by stimulating it. An increase similarly results when the hamstring muscles (the antagonists of the quadriceps) are relaxed by flexion of the knee.*

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* A very rare exception to this rule will be mentioned when the functions of the brain are considered. Moreover the knee-jerk is abolished in some cases of tumour of the cerebellum, a phenomenon to be remembered in connection with the fact that, while one part of the afferent impulses from the muscles acts on the cord, another part apparently passes up to the cerebellum. See the account of co-ordination of movement on the next page.

Co-ordination of Movement.—The co-ordination of muscular contractions, which results in the ordination or ordering or arrangement of movement, has been already described (p. 7), so far as its general features are concerned. We cannot ascribe to the spinal cord any process that can be designated the arrangement of movement; since the muscular contractions correspond to the activity of the motor cells of the cerebral cortex, and to the corresponding impulses along the pyramidal fibres. These determine the initial activity of the motor cells of the cord, both for the muscles that move a part and for those that oppose the action of the others and give steadiness and uniformity to the movement. But these impulses from the brain descend on spinal cells that are not in a passive, inactive, functionless state. Such a state probably never exists, for even when there is no voluntary activity there must be the activity on which the tone depends,—that, as we have just seen, must exist in all postures of the limbs. This state of the cells preceding voluntary activity is determined by the afferent impulses, chiefly by those from the muscles. Through these the muscles are adapted to posture, and in the mechanism for adaptation to posture we may trace also a mechanism for part at least of the co-ordination effected in the spinal cord. This seems to consist of two parts. (1) An automatic variation in the state of muscles, permitting the ordered performance of intended movements. The state of the afferent impulses acting from the muscle on the motor cells must be at once altered by the effect of the voluntary activity of the muscles, since this will alter the impulses in the muscle-nerves by the changed and changing pressure and tension on the tissues in which these nerves end—the pressure of the contracting and widening fibres and the tension in the antagonists that oppose the motion, but do not prevent it, and are made more tense by the muscles they are opposing. These impulses pass to the cord, and there act on the motor cells, and influence their state in a way that we can at present only guess at, but which must be of the utmost importance. There must of necessity be a definite relation between certain states of the muscle and the strength of afferent impulse produced thereby. If we conceive that an increase in the impulse (making it greater than corresponds to the degree of activity of the motor cells), such as must result from the increased tension on the muscle, lowers the activity of those cells, we can understand the way in which the contracting antagonists relax to permit movement which tends to occur. As soon as the resistance begins to yield, the tension-stimulus will be increased, and the total afferent impulse become disproportionately great in relation to the degree of activity of the cells; as a result, this activity is lessened and the muscles yield. This involves a constant regulation of the activity of the various motor cells by the afferent impulses. A like but opposite effect must be produced on the cells of the muscles producing the movement. This effect on the cells will, of course, blend with that of the voluntary impulse from the brain that is causing the movement. Thus the spinal muscle-reflex process permits the desired movement to take place, as it could not do if the state of the cells were not thus automatically regulated. (2) Relations between the various reflex processes are doubtless established by the repetition of movements, and thus planned mechanisms, as it were, are established in the cord; within their range the various movements occur. The state of the muscles in standing, for instance, is almost perfectly reproduced by the extensor spasm that occurs when the muscle-reflex processes are in great excess. Thus we have a mechanism by which a relatively simple voluntary impulse may produce an extensive co-ordinated movement. But what we can discern shows that we must regard spinal co-ordination as essentially a reflex process, chiefly due to the impulses from the muscles. The correctness of this view can hardly be doubted when we come to see its perfect harmony with the phenomena and the conditions of spinal inco-ordination.

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* See Sherrington, 'Brit. Med. Journ.,' 1893, vol. ii, p. 685.

Still it must be always remembered that this spinal process is of the nature of a subordinate mechanism, determining the precision of form rather than the actual arrangement and order of the muscular contraction. This is done by a higher process, a co-ordination which consists in the form of action of the motor cells of the cerebral cortex that is produced by still higher volitional processes. These, however, as we shall see, are themselves subject to a like influence and the seat of an analogous regulation, chiefly, it is probable, by impulses proceeding from the cerebellum, which are also due to the varying excitation of the muscle-nerves. That the nerve-fibres which pass up the posterior median columns conduct impulses from the muscles that determine cerebral co-ordination scarcely admits of doubt. Interruption of this path in any part of its course causes a defect of co-ordination, which resembles that due to cerebellar disease with sufficient closeness to add weight to other facts suggesting that it is through the cerebellum that the impulses carried by this path act on the cortex, regulating and determining the activity of its cells and the resulting movement.[*] The impulses may reach the cerebellum, as we have seen, from the grey matter of the post-pyramidal nucleus in which these fibres end.[†] But this primary co-ordination is not a function of the cord, although it may be lost in consequence of disease of the cord. The direct cerebellar tract may constitute a similar path, but of this we have only the evidence of destination and analogy.

It is probable that the mechanisms just alluded to, as constituted by the functional relations between the motor and sensory structures in the cord, very much facilitate the execution of many movements, and especially of those that are the more simple in their character. The less variation a movement is capable of, or needs, the more largely may the spinal mechanism be concerned in its arrangement; and hence such actions as standing and walking may be effected through a comparatively small number of the pyramidal fibres, and, as we shall see, they may still be performed when there is disease that would prevent a more elaborate action. It is also probable that in animals such spinal mechanisms are developed to a greater extent than in man, and care must be taken in drawing inferences from one to the other.

The impulses from the muscles that determine the muscle-reflex action and the spinal co-ordination, must pass by nerves that end in the grey matter and come into relation with nerve-cells, at or near the level at which they enter the cord. We do not know whether it is through these fibres, or through those that pass upwards without interruption, that the impulses are conveyed which, when excessive in degree, give rise to muscular sensations. But attention may again be called to the important evidence, afforded by such sensations, of the abundance of upward impulses of which we are normally unconscious. These nerves, it will be remembered, begin in the connective tissue of the muscles, and probably also in the muscle-spindles, and the acute pain of cramp, and the muscular tenderness in such affections as multiple neuritis, afford indirect evidence of impulses which we do not usually observe, and might almost doubt; while the manner in which such pain is excited is very clear evidence of the sensitiveness of the nerves to the modes of stimulation that have been assumed as effective. We have already seen (p. 17) that it is through such unfelt impulses that we probably derive the chief part of our conceptions of posture, a subject that must again be considered in connection with the brain.

INFLUENCE ON NUTRITION.—The nutrition of the *muscles* is under the control of the anterior grey matter, and probably of the motor nerve-cells. The influence is exerted through the motor nerves, and indeed consists in the normal integrity of the fibres (see p. 29).

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[*] This has been experimentally confirmed by Bechterew, who finds that section of the posterior columns, in various animals, above the lumbar enlargement causes a defect of co-ordination similar to that produced by loss of the cerebellum (Bechterew, 'Wjestnik psich. i. nevropat.,' 1889, vii, 1; and 'Neur. Centralbl.,' 1890, p. 82.)

[†] A connection since confirmed by Brosset, 'Des Connexions du Cervelet,' Paris 1891.

The nutrition of the *bones* and *joints* is also under the control of the cord, and probably is exerted through the posterior roots. It is true the growth of the bones is hindered when the anterior cornua are diseased, but no atrophic change seems then to occur in the osseous structure, and the influence of the paralysis may be indirect. A true trophic change, and an alteration in the joints, may occur in locomotor ataxy, in which the disease is of the posterior nerve-fibres.

The nutrition of the *skin* seems also to depend on nerves that have their course in the posterior roots. The clearest fact is that irritation of the nerve-structures has far more effect than simple loss of function. It is when the cord or nerve-roots are the seat of irritative inflammation that the most acute trophic changes occur, while mere degeneration of these nerves induces slower and slighter changes (see p. 28).

In a dog in which the spinal cord below the cervical region was removed piecemeal, trophic changes in the skin followed the section, but not the subsequent removal. It is hence doubtful whether trophic fibres actually take origin (*i. e.* have "centres") in the cord; in all cases it is difficult to exclude the effects of the fall of blood-pressure.*

Visceral Control.—Although the viscera are under the immediate control of the sympathetic system of nerves, they are related to centres in the spinal cord, and it is from these centres that the controlling influence is really derived, probably by means of the finer fibres of the anterior roots. The relation is the most direct and important, so far as concerns the diseases of the spinal cord, in the case of the organs over which the will has an influence, the *rectum* and the *bladder*. The centres for these are in the lumbar enlargement, but we do not know their exact position. They are probably complex reflex centres. We can best understand their action by studying them when voluntary influence is lost.

The centre for the sphincter ani is the more simple, but the system of action of each is probably similar. In the wall of each viscus we have muscular fibres to expel the contents, and at the mouth a sphincter arrangement to prevent continuous evacuation. Fæces or air in the rectum, and urine in the bladder, may excite the lumbar centre, and cause two effects—contraction in the wall and relaxation of the sphincter. This process can be controlled by the will to a considerable extent, although we are still ignorant of the precise mode in which the voluntary influence is exerted. But if the volitional path in the cord is interrupted above the lumbar centres, the will can no longer control the reflex processes; as soon as fæces irritate the rectum they are expelled by the reflex mechanism; as soon as a sufficient quantity of urine accumulates in the bladder a reflex contraction of the detrusor and relaxation of the sphincter cause its escape. The affection of the voluntary path for the sphincter is not always proportioned to that for the legs. If the damage to the cord involves also the sensory tract, the patient is unconscious of the action of the bladder or bowel. If the sensory tract is unaffected, the patient is aware of the process, but cannot control it. It is often said that there is permanent relaxation of the sphincters, but this is true only when the lumbar centres are inactive or destroyed. In this condition evacuation occurs as soon as fæces or urine enter the bowel or rectum. The urine escapes continuously, instead of being expelled at intervals. The condition is less obvious in the case of the rectum, because there is no such continuous passage of fæces into the rectum as there is of urine into the bladder. We may, however, distinguish between the two states of the rectum by the introduction of the finger. If the lumbar centre is inactive, there is a momentary contraction, due to local stimulation of the sphincter, and then permanent relaxation. If, however, the reflex centre and motor nerves from it are intact, the introduction of the finger is followed, first by relaxation, and then by gentle, firm, tonic contraction. I have verified this by introducing an india-rubber cylinder instead of the finger, and registering the pressure on the cylinder by connecting it with a recording apparatus. The relaxation is then found to be preceded by a very slight brief contraction, and

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* Goltz and Ewald, 'Arch. f. d. gesammt. Physiol.,' 1896.

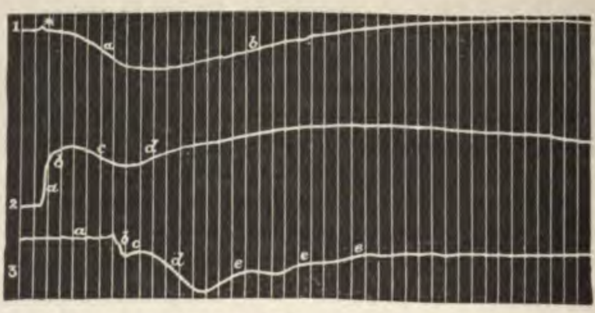

FIG. 85.—TRACINGS OF THE ACTION OF THE SPHINCTER ANI.

The vertical lines represent seconds of time.

1. Effect on contraction of sphincter of the injection into the rectum of a small quantity of air at *. *a*, fall in pressure due to the inhibition of the contraction; *b*, rise due to the slowly returning contraction. 2. Effect of the introduction of the instrument. *a*, sudden rise of lever at moment of introduction, due to the exposure of the instrument to the pressure of the sphincter (the top of this line represents the degree of previous contraction); *b*, initial rise due to increased contraction; *c*, fall from partial inhibition; *d*, subsequent contraction, rising to a greater degree than the initial contraction, and subsequently falling slightly. 3. Effect of cough. *a*, pressure of tonic contraction of sphincter (the slight irregularities are due to pulse-waves); *b*, fall in pressure, due to the movement of the instrument by the cough; *c*, initial contraction; *d*, relaxation of inhibited sphincter; *e*, *e*, *e*, rhythmical variations in subsequent rise. (From the 'Proc. Royal Society,' 1877.)

to be followed by unbroken tonic contraction. The relaxation may also be readily produced by any impression on the mucous membrane above the sphincter. Fig. 85 shows some of the tracings obtained.

The action of the bladder mechanism can be best understood by assuming that the motor centre really consists of two parts, one (MS, Fig. 86) maintaining the contraction of the sphincter, the other (MD) exciting the contraction of the detrusor fibres, and that these two parts are antagonistic; when one acts the other is inhibited. Thus, in normal rest, the sphincter centre is active, the detrusor at rest.

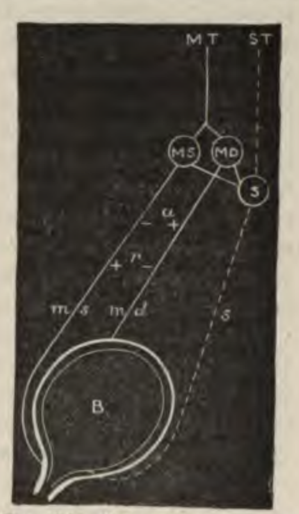

FIG. 86.—DIAGRAM SHOWING PROBABLE PLAN OF THE CENTRE FOR MICTURITION.

MT, motor tract; ST, sensory tract in the spinal cord; MS, centre, and *ms*, motor nerve for sphincter; MD, centre, and *md*, motor nerve for detrusor; *s*, afferent nerve from mucous membrane to S, sensory portion of centre; B, bladder. At *r* the condition during rest is indicated, the sphincter centre in action, the detrusor centre not acting. At *a* the condition during action is indicated, the sphincter centre inhibited, the detrusor centre acting.

Apparently, the process of micturition is effected by the automatic contraction of the wall, when the sphincter is relaxed. The sphincter is certainly under the influence of the will, which is able to maintain and increase its contraction, but it is doubtful whether the mere cessation of the voluntary influence is alone the cause of its relaxation. The elements of the process can be best discerned when it is weakened by slight disease. It is then clear that, in addition to the cessation of voluntary contraction, the relaxation is due to the sensory impulses from the mucous membrane at the neck of the bladder, but these do not seem to inhibit the spinal centre directly. This only becomes inactive when the afferent impulses act on the sensory centres in the brain, and it may be essential for the influence on these centres to be increased by the lowering of resistance involved in conscious "attention." If the sensation is not attended to no relaxation may occur. The effect may be, indeed, due to an automatic cerebral influence, maintaining the action of the spinal centre, which is only "taken off" when the sensory impression causes its full effect on the cerebral processes. The elements of the mechanism are scarcely to be discerned in health, but may under exceptional conditions be clearly perceived.

Although no other part of the *alimentary canal* is under voluntary influence, all parts are probably related to the spinal cord, by means of the connection between the sympathetic system and the nerve-roots. Constipation is extremely common in disease of the spinal cord, and is often greater than can be accounted for by the mere of the changes in nutrition, but it is probably not the sole mechanism by which these changes are produced.

There is a special connection between the sympathetic and the cervical region of the spinal cord. This region contains the path to the visceral nerves that control the sugar-forming functions of the liver. The lower part of the cervical enlargement contains also an important centre which controls the sympathetic nerves for the vessels of the head and face. The path of central influence for the radiating fibres of the iris passes also from the region of the third nerve nucleus, down the cervical cord, by the lowest cervical or first dorsal nerve, to the sympathetic, and then ascends the cervical trunk, and passes along the fibres which accompany the internal carotid artery to the nasal division of the fifth cranial nerve. In this the fibres run for some little distance, ultimately reaching the dilator muscle as the long ciliary nerves. Fibres also pass from the cervical cord to the sympathetic, through which the action of the heart is accelerated.

MUTUAL RELATION OF THE FUNCTIONS OF THE SPINAL CORD.—It may be well to present, in the form of a table (p. 252), the relation of the motor and reflex functions of the cord, as far as our knowledge of these relations extends. Many points are still uncertain. The sensory relations will be sufficiently obvious from the figures. The table does not need any detailed explanation. It is merely a comparative collection of the facts that have been stated in the preceding pages. We shall find the value of this collocation of functions when we consider the effects of transverse lesions of the cord at different levels.

loss of power of the abdominal muscles. The pain in the back that is so common in disease of the stomach has been regarded as a reflected pain, due to this connection.

The *uterine* functions are probably independent of the cord in greater degree than those of any other organs under the influence of the sympathetic. The function of menstruation goes on independently of cord disease, and may be regular although there is absolute arrest of all conduction, sensory and motor. Pregnancy and labour may proceed in normal course, in spite of such disease of the cord as causes complete motor paraplegia.

Sexual Functions.—The activity of the sexual organs depends on the integrity of the reflex loop to and from a special centre, also situated in the lumbar enlargement, but the due action of this centre depends on cerebral (psychical) as well as on reflex influences. Disease of the centre, or of the nerves leading to or from it, abolishes sexual action. The sexual reflex is, however, one of the cutaneous reflexes, and it shares the condition of these rather than of the muscle-reflex processes. It has been stated that in tabes loss of sexual power is associated with loss of tactile sensibility of the glans penis.*

The centre is probably double, and its action is impaired by interference with either half. When, by disease higher up, the connection with the psychical centres is interrupted, the sexual act cannot be perfectly performed. If the path from the controlling centre (p. 20) is unimpaired, the reflex sexual processes are not in excess, and may even be diminished; but if the path from this controlling centre is also interrupted, the reflex sexual processes are in excess like the other superficial reflexes, and priapism results. If the reflex centre, or connecting paths, are partially diseased, the sexual process may be impaired and imperfect, but not abolished.

Vaso-motor Centres.—The sympathetic nerves to the vessels are influenced from the spinal cord. It is probable that the path is by the fine fibres of the anterior roots, and that most of the constrictor fibres leave the cord between the third dorsal and the second lumbar, while the dilator fibres are more widely scattered, many arising in the upper dorsal region, while others leave the cord in its lumbar and sacral portions ("pelvic outflow"). Some facts of disease suggest that the subsidiary vaso-motor centres are situated in the intermediate grey matter; and this conclusion is supported by the important researches of Gaskell, which refer the function to the small cells of the intermedio-lateral tract, which he traces upwards to the vaso-motor centre in the medulla. The action of the vaso-motor nerves may be deranged by disease of the cord. It is probable that such a relation exists between all parts of the vaso-motor system of nerves and the cord, and that the relation obtains between each segment of the cord and the vascular nerves to the corresponding part of the trunk and limbs. This derangement in disease may take a part in the production of the changes in nutrition, but it is probably not the sole mechanism by which these changes are produced.

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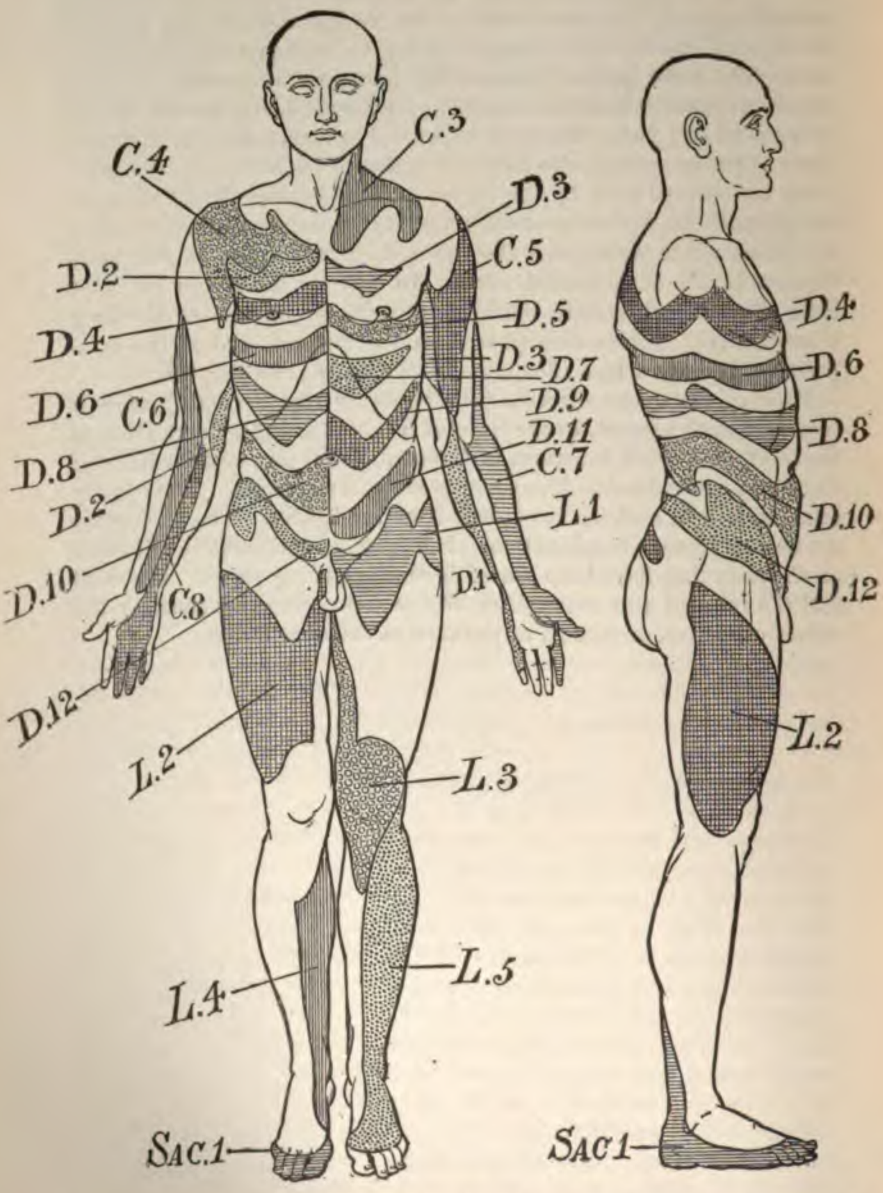
* See Brown, 'Lancet,' 1898.

There is a special connection between the sympathetic and the cervical region of the spinal cord. This region contains the path to the visceral nerves that control the sugar-forming functions of the liver. The lower part of the cervical enlargement contains also an important centre which controls the sympathetic nerves for the vessels of the head and face. The path of central influence for the radiating fibres of the iris passes also from the region of the third nerve nucleus, down the cervical cord, by the lowest cervical or first dorsal nerve, to the sympathetic, and then ascends the cervical trunk, and passes along the fibres which accompany the internal carotid artery to the nasal division of the fifth cranial nerve. In this the fibres run for some little distance, ultimately reaching the dilator muscle as the long ciliary nerves. Fibres also pass from the cervical cord to the sympathetic, through which the action of the heart is accelerated.

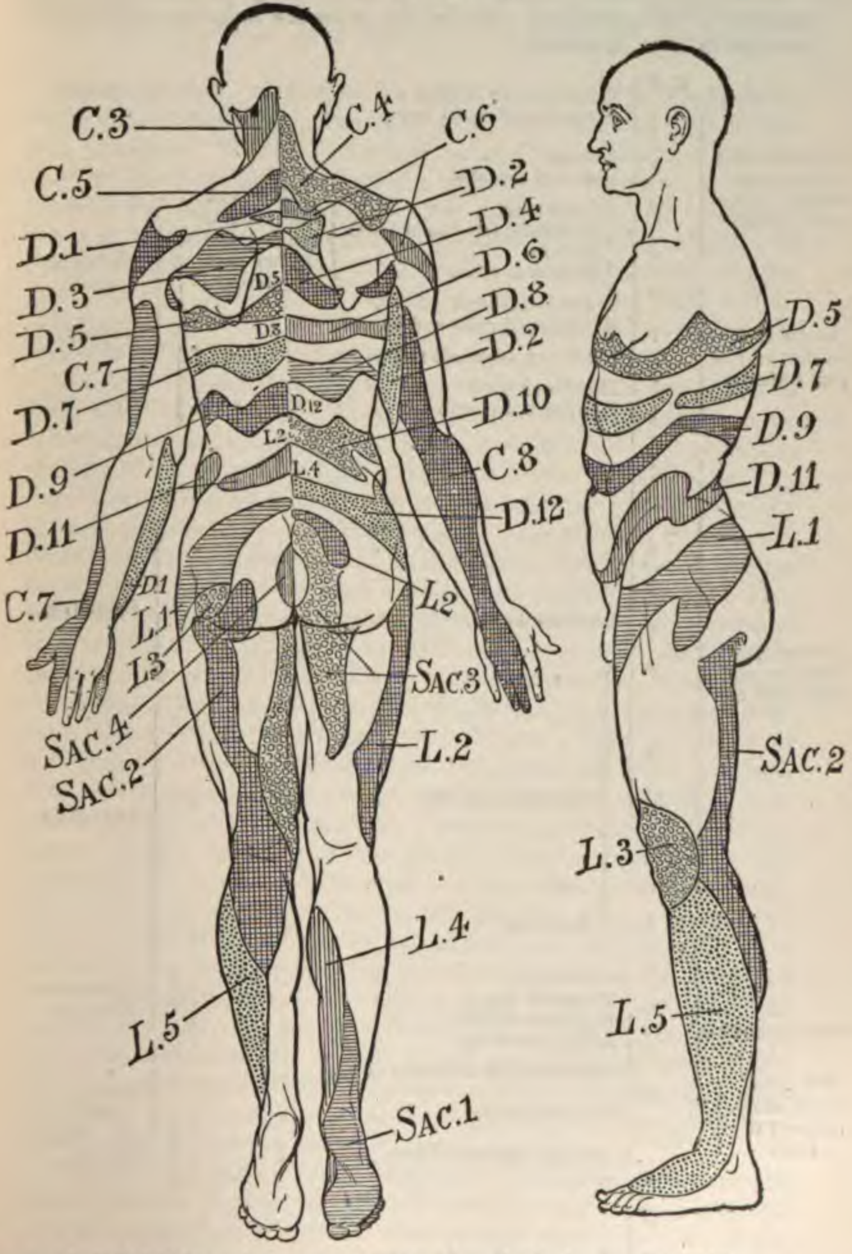
Mutual Relation of the Functions of the Spinal Cord.—It may be well to present, in the form of a table (p. 252), the relation of the motor and reflex functions of the cord, as far as our knowledge of these relations extends. Many points are still uncertain. The sensory relations will be sufficiently obvious from the figures. The table does not need any detailed explanation. It is merely a comparative collection of the facts that have been stated in the preceding pages. We shall find the value of this collocation of functions when we consider the effects of transverse lesions of the cord at different levels.

Fig. 87.



Figs. 87 and 88.—Dr. Henry Head has been kind enough to permit the use of these. researches, mainly on cases of herpes zoster and of localised spinal cord lesions. It majority are approximately so, and, in any case, they will serve as a substantial

Fig. 88.



They were devised by him, and the sensory areas are marked out as a result of his Is not claimed that all the areas are absolutely correct. It is believed that the basis for future research.

Table showing approximate relation to the spinal nerves of the motor and reflex functions of the spinal cord. The sensory areas are indicated with sufficient clearness in Figs. 87 *and* 88.

				REFLEX.
	C. *1*	Small rotators of head. Depressors of hyoid bone.		
Sterno-mastoid, upper neck muscles, upper part trapezius	*2*			
	3	Scaleni. Lev. ang. scapulæ.		
	4	Diaphragm Pectoralis (clavicular part)	**Deltoid and other shoulder muscles, and sternal part of pectoralis**	**Dilatation of pupil.**
	5	Serratus Flexors of elbow		**Scapular.**
Lower neck muscles, middle part trapezius	*6*	Pronators, triceps Extensors of wrist and fingers		
	7	Flexors of wrist and fingers		
	8	Muscles of hand Extensors of thumb		
	D. *1*	Intercostals		
Lower part trapezius and back muscles	*2*			
	3			
	4			**Epigastric.**
	5			
	6			
	7	Abdominal muscles		
	8			**Abdominal.**
	9			
	10			
	11			
	12			
	L. *1*	Ileo-psoas		
Lumbar muscles	*2*	Cremaster Flexors of hip		**Cremasteric.** ***Knee-jerk.***
	3	Extensors of knee Adductors of hip		
	4	Extensors and abductors of hip		**Gluteal.**
Peroneus, flexor of ankle, extensor of ankle	*5*	Flexors of knee		***Foot-clonus.***
	S. *1*	Intrinsic muscles of foot		**Plantar.**
	2			
	3	Perinæal and anal muscles.		
	4			
	5			

SYMPTOMS OF DISEASE OF THE SPINAL CORD: INDICATIONS OF THE SEAT OF THE LESION: ANATOMICAL DIAGNOSIS.

The symptoms of disease of the spinal cord consist in derangement of its various functions; the loss of some, the exaltation and perversion of others. We have already considered these functions, and the parts of the cord by which they are subserved. We may now briefly consider the general character of their derangement, and its significance in regard to the position of the disease. The combination of symptoms indicates the seat of the lesion; we infer its nature from their mode of development, and other considerations, which will be subsequently discussed. It is always important to keep these two elements of the diagnosis distinct in the mind.

A common feature of the symptoms of disease of the spinal cord is their bilateral character. This depends on two causes, of which, however, only the first is peculiar to the cord, and can be regarded as the special cause of this feature of its diseases. The structures in each half of the spinal cord are in such close proximity, that any random process, such as hæmorrhage or inflammation or pressure, readily affects both halves—can, indeed, hardly fail to do so in some degree, and usually to such an extent as to cause the bilateral character of the symptoms to be obtrusive. Secondly, morbid processes that commence in the nerve-elements and affect them according to their function ("system diseases") usually involve the corresponding structures of the two sides, and the spinal cord contains many structures that are liable to such processes. Hence a large proportion of the diseases of the spinal cord involve both sides and cause symptoms that have a corresponding range. The leading fact that results from this is, that the characteristic type of palsy is "paraplegia." The word means literally paralysis of the parts beneath or beyond a certain locality, and involves the distinction of a motionless part of the frame, which no doubt arose from the variable extent of this, in comparison with the uniformity of the region affected in "hemiplegia." But the second condition causing the bilateral character of the symptoms is shared equally with those outlying parts of the spinal cord—the peripheral nerves; and as this condition obviously determines a stricter symmetry than does the first, it follows that this character is not, alone, of much significance, and is indeed least significant when most complete. In the affections that are strictly symmetrical, therefore, other features must also be taken into account.

Another character of spinal symptoms, not shared by those of the nerves, consists in their variation in vertical extent according to the position or extent of the lesion. But we must distinguish two classes of symptoms within the region in which they exist. (1) Those which depend on the interruption of the conducting path to or from the brain, and involve the whole region below the disease. (2) Those which depend on damage to the central structures in the cord and nerve-roots, which are present only at the level of that damage, *i. e.* in the parts functionally related to that part of the cord. The two sets of symptoms may be distinguished as "conducting" and "central;" but in many diseases those at the level of the lesion are chiefly due to the irritation of the nerve-roots, and these are most usefully designated "root symptoms."

Disease of the cord does not always cause bilateral symptoms. A little consideration will show that this must be so. A random process, of small extent, may damage one side only. It may affect one or several structures on one side or all of them. Nevertheless it is very rare for such a process to spare the other side altogether, and practically no lesion affects all parts of one side without damaging, in some degree, the other side, at any rate for a time. In very rare forms of traumatic lesion, however, the damage to the other side is almost imperceptible. Again, "system diseases," in quite exceptional cases and for unknown reasons, are limited to one side; much more frequently they may affect one side earlier and more than the other. Such unilateral symptoms indicate a condition awkwardly termed "hemi-paraplegia."

MOTOR SYMPTOMS.—*Loss of motor power* results from interference with the motor path in any part of its intra-spinal course,—pyramidal tracts, anterior grey matter, anterior nerve-roots. We have seen that the path may be divided into two segments, upper and lower (see p. 213). The spinal cord contains a considerable portion of the nerve-fibres of the upper segment, and their lower termination in the grey matter, but only the commencement of the lower segment, the motor ganglion-cells, and root-fibres proceeding from them. The chief part of the lower segment is outside the spine, in the nerve-trunks. So far as the loss of power is concerned the effect is the same, in whatever part of the motor path the interruption is situated; but the other symptoms that accompany the loss of power differ very much according as the interruption is in the upper or lower segment. The lower segment influences muscular nutrition, and forms part of the path of reflex action. Hence, as we shall presently see, the muscles waste, and reflex action is lost, when this is diseased; but when the upper segment is damaged there is no muscular wasting, and reflex action, instead of being lost, is commonly increased. *The symptoms produced by disease of either segment are essentially the same, whatever part of the segment is diseased, whether the cell at the upper extremity, the fibre, or the ramification at the lower extremity of the fibre.* If, for instance, the intra-muscular nerve-endings are paralysed by curara the symptoms produced are the same as if the motor fibres in the nerve-trunks are divided or their cells in the cord are destroyed. Disease of the termination of the upper segment in the grey matter of the cord must, of necessity, produce the same effect as disease of the pyramidal fibre itself, or the cell in the cerebral cortex from which the fibre springs. We shall afterwards see that this consideration has very important applications.

If a fibre of either segment is seriously damaged, it degenerates below the division, since its nutrition depends on the influence of the cell from which it has sprung. If damaged only by pressure, however complete may be the evidence of degeneration, regeneration is possible, even after one or two years. It is possible that such regeneration also occurs when the damage is by inflammation. The nutritional stability of the fibre, or rather of its essential element, the axis-cylinder, becomes less, and more easily deranged, the greater the distance from the parent cell, and it is least in the terminal ramification of each segment. This is probably the reason why curara acts chiefly on the intra-muscular nerves, and it explains the influence of many poisons on the peripheral nerve-endings, and the facts of nerve-degeneration in tabes and multiple neuritis, &c. The law is probably also true of the upper segment; and if so, many facts of pathology become clearer to us, as will be seen in the chapter on spastic paraplegia. It may thus be taken as a general law of the utmost importance that *the vitality of the processes of a neuron and their power of resisting morbid influences diminish in proportion to their distance from the cell body of which they are really part.*

A lesion of the pyramidal tract causes loss of power in all parts below the level of the disease—that is to say, of all parts the fibres for which are interrupted. A lesion of the grey matter, or of the anterior roots, causes paralysis only of those parts which are functionally on the level of the lesion. The two mechanisms often coincide. A transverse lesion in the cervical enlargement, for instance, may cause paralysis of the arms from the damage to the grey matter and nerve-roots, and paralysis of the legs from damage to the pyramidal fibres. The disease involves, primarily in the former case, the commencement of the second segment of the motor path for the arms; in the latter, the middle of the upper segment. The associations of the paralysis differ accordingly. The parts affected by a lesion at any level will be readily ascertained by an examination of the table of functions on p. 252.

It is important to note that the affection of motor power is often incomplete. It may then involve one set of muscles more than another. The flexors or the extensors may be chiefly paralysed. In disease of the dorsal cord, it is very common for the flexor muscles of the hip and knee to suffer more than the extensors. Why this should be we do not know, but the fact is important, because considerable weakness of the flexors of the knee is readily overlooked. In examining motor power each set of muscles should be separately tested.

Motor over-action; Spasm is frequent in disease of the spinal cord and its membranes. It may present the form of *tonic* spasm, which when persistent is termed *rigidity;* or of *clonic* spasm, which is usually transient and paroxysmal. Tonic spasm, persistent, and involving only a certain group of muscles, causes distortion of the parts to which they are attached, and is often termed *contracture.*[*] Tonic spasm, paroxysmal or persistent, is far more frequent than clonic spasm, which occurs chiefly in association with tonic spasm as the muscular clonus described at p. 22, or as a consequence of certain functional derangements. Tonic spasm may be the result of direct irritation of the motor structures, or may depend on over-action of the reflex centres, due not to irritation, but to deficient control. Either form may be acute or chronic. The acute irritation which causes spasm is generally inflammation of the membranes, rarely an acute lesion of the substance of the cord. The chronic irritation is chiefly due to compression of the motor fibres—either of the pyramidal tracts, when there may be persistent unvarying spasms in the parts below the disease, related to the fibres irritated,—or of the nerve-roots, when similar spasm is produced in the parts at the level of the lesion. The spasm that depends on reflex action is seen chiefly in the legs and trunk, but especially in the former. It may preponderate in either the flexors or extensors, so that the legs, when rigid, may be drawn up or straight out. The flexor spasm seems to be due to an over-action of the centres for cutaneous reflex action, the extensor spasm chiefly to that of the centres for muscle-reflex action, although it may also be excited indirectly by a cutaneous impression. This form will be considered in connection with the reflex over-action.

Muscular Contraction.—The actual shortening of muscles, by which they cannot be passively extended to their normal length, is due to tissue changes fixing the active persistent tonic spasm or contracture just mentioned. The rapidity with which it occurs depends on the uniformity of the spasm, and we may distinguish three modes in which it arises, which differ in the time required and degree attained. (1) When one set of muscles is paralysed, their opponents, never being extended, quickly become fixed in the contracted condition which they assume in virtue of their power of adaptation to posture. Thus in palsy of the flexors of the ankle, the calf muscles quickly become contracted, so that the foot cannot be flexed on the leg even up to a right angle. The same thing happens by a similar mechanism, when one posture is unceasingly maintained and one set of muscles is never extended, *e. g.* persistent flexion of the knee leads quickly to structural shortening of the hamstring muscles. (2) Where there is unchanging active contracture from chronic irritation of the motor fibres by a growth, or permanent reflex contracture, as when reflex flexor spasm keeps the hip and knee permanently flexed, similar structural changes occur, so that passive elongation soon becomes impossible. (3) In paroxysmal tonic spasm of intense degree, if one set of muscles is stronger than their opponents, their action may so preponderate as to lead to a greater active contracture than their opponents present, and permanent shortening may occur; although it does so slowly and to a slighter degree than in the other forms because the muscular action is more or less paroxysmal and varying, and the muscles are at times extended by their opponents. Thus in paroxysmal extensor spasm in the legs, no shortening occurs in the muscles moving the knee, because they are equally balanced, but the calf muscles, being stronger than the flexors of the ankle, contract the more, and extend this joint, whenever a paroxysm of spasm comes on, and, after a time, may undergo slight structural shortening, so that the foot cannot be flexed on the leg beyond a right angle.

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[*] The reverse of this condition, loss of muscular tone permitting unusual freedom of passive movement, is described in tabes under the name of *hypotonie musculaire* Frenkel).

It is of great importance to distinguish the shortening of muscles due to tissue changes in them, from the active contracture that may closely simulate it. In the latter, gentle extension, kept up for a few minutes, restores the muscle to its normal length. The importance of the distinction is due to the fact that the structural shortening can be removed by tenotomy or forced extension, while these measures are useless in the case of active contracture, which would subsequently reassert itself.

Tremor is a symptom which should be mentioned, for it is at least associated with disease of the spinal cord in insular sclerosis (see Vol. II). It also occurs in degenerative diseases, such as paralysis agitans or general paralysis, and as the senile tremor of old age. In these conditions it probably depends upon changes in the cerebral cortex. It is characteristically present in toxic conditions manifesting themselves chiefly in affections of the peripheral nerves, such as those arising from alcohol, lead, and mercury. The spontaneous twitchings of muscular fibres known as "fibrillary twitchings" occur especially in association with degenerative disease of the anterior cornual cells.

Inco-ordination of movement, although motor, depends usually on disease of afferent fibres. Its characters have been already described in outline (p. 10).

It occurs in three forms, of which the third differs entirely from the others in aspect and nature, and alone is a true motor symptom. The two chief forms are the following:—(1) Simple disorder of voluntary muscular contractions, which, when considerable, amounts to a wild irregularity of movement, and when slight is only manifested by slight irregularity when the guidance of vision is withdrawn—as in the inability to maintain the muscular contractions in the perfect uniformity needed for equilibrium when the base of support is rendered small by the juxtaposition of the feet. The delicacy of the test is increased by the removal of the firm base afforded by the boot, and by closure of the eyes. The increase in unsteadiness when the eyes are closed is sometimes termed "Romberg's symptom." This condition results from interruption of the afferent path from the muscles to the spinal cord, including the fibres that ascend the cord and those that subserve the muscle-reflex process at the level of entrance. The effect of the loss of the latter is a cessation of the chief part of spinal co-ordination, which, as we have seen, depends on those processes, co-operating with the voluntary impulse. Hence it is associated with loss of the knee-jerk. The loss of other forms of reflex action does not seem capable of causing inco-ordination. In complete interruption of the root-fibres from the muscles the mechanism of the next form is necessarily conjoined. (2) Inco-ordination resembling the slighter degrees of that just described, and consisting especially in such a defect of equilibrium as results from disease of the middle lobe of the cerebellum, is produced by disease of the posterior median columns above the lumbar enlargement, or, if it extends into the latter, leaving the root-zones free so as not to impair muscle-reflex action. This is apparently produced by interruption of the path by which impressions from the muscles are conveyed to the posterior nuclei of the medulla, and probably thence to the cerebellum (p. 244). The effect is to deprive the motor cortex of the guidance afforded by these impulses. The difference between this form and the slighter degrees of the first is that the muscle-reflex action is not lost, the knee-jerk being preserved. They are both spoken of as *ataxy* also. (3) Irregular compression of the motor fibres in the cord apparently causes the peculiar jerky inco-ordination seen in disseminated sclerosis, which is supposed to depend on unequal and irregular conduction along the fibres, due to their compression. This form, however, needs further study.

SENSORY SYMPTOMS.—Loss of sensation is a very common effect of disease of the spinal cord. It may be total, and involve all forms of sensibility, or partial, and affect only some forms. The statements made on pp. 13—19 regarding impairment of sensation generally, and the method of ascertaining it, apply to that which results from cord disease, and need not be here repeated. It has been also pointed out (p. 78) that loss of sensation occurs less readily than loss of motor power. In cord disease it is common for muscular paralysis to exist with intact sensibility. This may partly depend on the fact, ascertained by experiments on the effect of local anæmia of the cord, that the sensory fibres, of the nerve-roots at least, seem to have more resisting power than the motor fibres. Sensation may be impaired by disease of any part of the sensory path—posterior roots, probably also the posterior cornua and commissure, or the conducting tracts up the cord. A division of the sensory path into upper and lower segments is conceivable, analogous to that of the motor path, although we have not the same clear ground for the distinction into simple segments of similar composition. Histological researches show that most of the centripetal neurons have short axis-cylinders, and suggest that upward conduction is subserved by a number of relays.

Disease of the posterior nerve-roots causes loss of reflex action as well as impairment of sensation, just as disease of the anterior roots interferes with reflex action as well as with motor power. Interruption of the sensory conducting tracts higher up leaves reflex action unaffected. But a focal lesion, such as transverse myelitis, may damage both the conducting tracts and the nerve-roots at the level of the lesion, or may affect only one of these. Disease outside the cord, compressing it, may have the same double effect. Hence it is important to test sensation at the level of the lesion, as well as in the parts below, and to remember that the "level of the lesion" may involve the limbs or the trunk. Areas of anæsthesia may thus be found on the trunk when there is none on the legs, and may be of considerable diagnostic importance, *e.g.* in spinal caries.

Random disease of the nerve-roots outside the cord usually impairs all forms of sensibility, although slight damage may arrest the conduction of tactile impressions, and not those of pain, which are probably more energetic. Disease of the roots within the cord often causes only partial loss, because the fibres which conduct different impressions have a different course. But loss of one form of sensibility from disease of the root-fibres or nerve-fibres usually depends on the kind of disease, and is especially due to degenerative changes, the result of some present or past local influence. Disease higher up the cord still more frequently causes partial loss; either sensibility to pain or to touch may be impaired. That to temperature is rarely affected without that to pain. We do not yet know precisely the significance of this special form of loss, because, as we have seen, we are still uncertain as to the path for each form of sensibility. It is highly probable, however, that loss of sensibility to pain is produced by disease of the grey matter immediately anterior to the postero-median column.

Disease of the posterior median column, and possibly that of the direct cerebellar tract, certainly impairs the conduction of impressions from the muscles. It probably abolishes the "muscular sense" of posture and movement, although this does not entail a distinct sensory loss. But there may be also a greater loss of muscular sensibility, so that the normal sensitiveness to pressure and passive extension (and even at last to electrical stimulation) may be impaired or lost. The "common sensibility" of muscles is probably lost only when the disease of the nerves is greater than suffices to abolish what is termed the "muscular sense." Interference with this path in the nerves or roots outside the cord abolishes muscle-reflex action, and also causes more pronounced symptoms (ataxy, &c.) than disease higher up the path within the cord.

Increased sensitiveness, hyperæsthesia and hyperalgesia, are also common in disease of the spinal cord, and usually depend on irritation of the conducting fibres in some part of their course. Probably the irritation produces the effect by intensifying the impulse as it passes, since the phenomena of stimulation of nerves show that their axis-cylinders have some power of transforming other forms of energy into

[illegible] force, i.e. of evolving nerve-force, and therefore of increasing
strength of that which passes along them.
The term "hyperæsthesia" is commonly used in the sense of
hyperalgesia." In the strict sense of the word, hyperæsthesia is
[illegible] observed or even searched for. When the sensibility to touch
"increased" there is some perversion of the sensation, not a simple
[illegible] The sensation may be felt as "thrilling" or "shock-like
[illegible]" When there is an extreme increase, pain may be pro-
[illegible] by a touch, but it is more probable that the touch stimulates
[illegible] nerves of common sensibility, than that actual pain
[illegible] through the tactile nerves. A touch may cause pain when
[illegible] as a touch, as in the condition termed "anæsthesia dolo
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[illegible] in the spinal cord. Impairment of sensibility for pain a
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More often they are dull pains, cl
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themselves and their medical attendants. The mistake is the more easily made, because other symptoms suggestive of spinal disease may be inconspicuous, and the rheumatoid pains in chronic cases may be influenced by weather, being much more troublesome in damp and cold than in fine and dry weather. In all cases, persistent rheumatic pains in the limbs should suggest the possibility of spinal disease, and watch should be kept for such symptoms as local loss of power, or alterations in reflex action. There is a third class of pains—resembling root-pains—which depend on degenerative changes in the nerve-fibres; the molecular alterations that result give rise to upward impulses of considerable intensity. They are met with in locomotor ataxy and in multiple neuritis, and may be dull as well as acute. The position in which these various radiating pains are felt—legs, trunk, or arms—depends upon the seat of the disease. Although often produced in the root-fibres, these pains may also be due to the peripheral nerves.

It is especially important to note that the root-pains are frequently felt as a sense of constriction, a painful sense of tightness, as if a band were tied tightly around the part—the "girdle-pain," as it is called. When there is transverse damage to the cord, at the lowest part of the healthy region there is a state of irritation of the sensory nerves, and this irritation (referred to the nerve-endings) causes the girdle-pain. It is named from its frequency at the level at which a girdle is worn, the middle of the trunk, which depends on the dorsal region being the most common seat of disease; but it may be felt lower down, about the groins or even the legs, or higher up, in the arms. In the limbs it is a mere sense of tightness.

The girdle-pain is a symptom chiefly of inflammatory and degenerative processes within the cord. Pressure on the nerve-roots usually causes acute pain, but the most severe root-pains are those met with in disease of the bones of the spine. These have also, more than any other kind, the characteristic that they are increased by movement, and in growths commencing in the bones (which are usually malignant) this feature is of considerable diagnostic importance. The suffering in such cases is so great as to have gained for the resulting symptoms the ominous designation of *paraplegia dolorosa*. The pain in this condition may have a localising value, that in carcinoma being frequently referred to the neighbourhood of the column, while in caries it seems more frequently to affect the side of the chest.

Spontaneous sensations, other than pain, are very common in disease of the spinal cord, and are often of considerable diagnostic importance, but suggestive rather of the presence of a morbid state than of its nature. They may present many varieties of character, but the familiar sensation produced by pressure on a nerve-trunk, when the part is said to be "asleep," or to have "pins and needles," is the most common and the most significant. It may occur in functional disturbance of the cord as well as in organic disease, and also is very

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[illegible] but it has at present no special significance as to
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[illegible] "numbness" is also common, and may occur, in
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the knee-jerk is present or is lost, on acecount of the readiness with which its occurrence may be prevented by inability to relax the muscles, and its presence may be simulated by a true reflex action (see p. 238).

Excess of reflex action implies, of necessity, the integrity of the reflex arc concerned, and shows that organic disease, if it exists, is higher up the cord. Each form of reflex action is often increased. In some acute diseases, as acute meningitis, and probably also in some chronic diseases, the increase may be due to an irritation of the centres, but in most forms of chronic disease it is apparently the result of a loss of control, and indicates disease between the centre concerned and the brain. We have already considered (p. 240) the probable mechanism, and have seen that the excess of the muscle-reflex action is related to disease of the pyramidal fibres, and especially to the loss of the terminal part of these fibres, within the grey matter, adjacent to the centres concerned.[*] This is the significance of considerable excess. Degeneration of the terminal portions of the fibres is commonly due to a descending degeneration of the fibres themselves. It is possible, however, that the degeneration of the endings of the pyramidal fibres may be primary, as is that of the nerve-fibres. (See Primary Spastic Paraplegia.) The increase of reflex action is chiefly manifested in the legs, in which reflex action is normally more active and important than in the arms. The increase in the cutaneous reflex action is shown by its occurrence on a slighter stimulation of the skin, and by a greater degree and extent of the muscular contractions produced. In these the flexors usually predominate, so that the leg is drawn up in a strong movement. As the action of the sexual organs depends, in part, on superficial reflex processes, priapism is common when the reflex excess is great. The increase of the muscle-reflex action is shown by an increase of myotatic irritability, and therefore of the so-called "tendon-reflex contractions,"—the increased knee-jerk, rectus-clonus, foot-clonus, wrist-jerk, elbow-jerk, jaw-jerk, jaw-clonus, &c. These have been already described. As already stated, the excess of this irritability is at first moderate, and slowly increases. A high degree of over-action is never suddenly attained, as it may be in the case of cutaneous reflex action. It would seem as though, in the centres liberated from control, a capacity for excessive action is slowly developed, which ultimately results in tonic spasm.

Not only is the common cause of this excess damage to the pyramidal tracts higher up the cord, entailing their secondary degeneration; but if such disease higher up—as, for instance, primary myelitis or injury to the cord—is followed by abolition of the muscle-reflex action in the parts below, it is the result of inflammatory damage to the lower part of the cord in which the centres are situated, generally by a descending myelitis. Sometimes the original myelitis involved the whole or a considerable part of the cord below, and the indications of a transverse lesion were merely those of the upper limit of the disease. It is not uncommon for the secondary myelitis below an original transverse lesion to succeed it, and abolish the reflex action which was at first unimpaired. I have several times met with such a sequence. A primary dorsal myelitis has left reflex action in the legs unimpaired and tending to excess, with the sphincter ani in its condition of uncontrolled automatic action, when after a few days, a week or more, indications of a recurrence of myelitis have been attended by abolition of all reflex action, including the knee-jerk, by flaccidity of the muscles, with rapid development of the reaction of degeneration in them, and by complete relaxation of the sphincter ani. This is the state from the first in the cases in which the primary myelitis involves the whole lower part of the cord. Sometimes, indeed, such descending inflammation is limited to the grey matter or to the posterior columns, with corresponding limitation of the symptoms, but with a similar abolition of reflex action. Of course, such descending inflammation is a very different thing from the secondary descending degeneration of the pyramidal tracts.[*]

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[*] It is interesting to note the analogy between the effects of degeneration of the termination of the two segments of the motor path. The increased activity of the muscle-reflex centres, which results from degeneration of the termination of the upper segment, presents some similarity to the increased voltaic irritability of the muscular fibres which results from degeneration of the termination of the lower segment.

Over-action may perhaps be also produced by a state of irritation propagated from above, as in the "early rigidity" of hemiplegia; we have seen that the motor impulses probably pass from the pyramidal fibres through part at least of the muscle-reflex mechanism. Primary over-action seems also to occur, although apparently it only attains a slight degree. It is probably the cause of the trifling excess met with in cases of defective nutrition of the spinal cord or of the nervous system generally.

Reflex spasm.—We have seen (p. 26) that the muscular state on which myotatic irritability depends, assumed to be due to a muscle-reflex action, is probably identical with physiological *tone*. Whenever there is a considerable increase in the irritability there is a tendency to tonic spasm, in which there is a balanced contraction of the muscles, fixing the limbs in the position of extension. At first there is merely slight stiffness of the legs, especially felt on waking from sleep; afterwards the legs, when extended, become distinctly stiff, although they may be quite supple when flexed. If gradually extended, when near full extension the spasm suddenly comes on and completes the movement, as the blade of a pocket knife moves under the influence of the spring. Hence this has been termed "clasp-knife rigidity." Ultimately the extensor spasm may be so great that, when it occurs, both legs are rigid, and are so firmly connected with the pelvis that if one is lifted from the bed the other is moved also. This is only met with in organic disease, and is an important diagnostic indication. Paroxysms of spasm may be brought on by any impression on the sensory nerves, a prick on the skin, or an attempt to obtain the clonus. The extreme spasm prevents any clonus being obtained, and it may be necessary to flex the limb before an attempt to obtain it is successful. As violent spasm is passing off, moreover, the tension of the muscles may alone set up a clonus, so that the paroxysm of tonic spasm passes into clonic spasm, a phenomenon that was termed by Brown-Séquard "spinal epilepsy," on account of the superficial resemblance to the sequence of spasm in an epileptic fit. This clonic spasm may often be arrested by any strong painful impression on the sensory nerves, a pinch of the skin, or painful flexion of the big toe; the strong stimulation of the nerves of common sensation inhibits the reflex centre concerned in the clonus. There is always much weakness of the legs when there is much spasm, and there may be almost complete paralysis. Hence the condition has been termed "spastic paraplegia." It is probable that the central reflex mechanisms concerned in the production of this extensor spasm are those that subserve the act of standing. The spasm often enables a paraplegic patient to stand, when his voluntary power is quite insufficient for the act. In advanced cases the extensor spasm may be varied by attacks of flexor spasm, which at first come on chiefly during sleep, but after a time preponderate, and may ultimately be permanent; this is always an unfavorable symptom, since, for some reason not yet understood, this flexor spasm indicates a condition of the spinal cord from which recovery is rare.

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[*] In consequence of the occurrence of this inflammation in the lower part of the cord having been overlooked, it has been thought that a transverse lesion higher up has the power of abolishing reflex action below, where the cord is not damaged except by descending degeneration. See, however, p. 272, note. In one case, however, described by Bruns ('Arch. f. Psychiatrie,' vol. xxv), the knee-jerk was absent although the grey matter was found to be unaltered. There was abolition of faradic irritability in the legs ascribed to œdema.

VASO-MOTOR AND TROPHIC DISTURBANCE.—*Changes in Nutrition.*—Considerable wasting of the *muscles* indicates disease of the motor nerve-cells, or of the nerve-fibres proceeding from these cells. If the disease is acute there is rapid degeneration of the nerves, with the reaction of degeneration in the muscles (see p. 71). The wasting in these cases is always great, and there is always loss of reflex action. Occasionally some fibres of the nerve and muscle suffer and others do not, causing the "middle" or "mixed" form of reaction (see p. 32). In rare cases, in which the nerve degeneration set up is of an intensely irritative character, the muscles may rapidly lose all irritability in consequence of true degeneration of the fibres. I have observed this, for instance, in a growth in the lumbar region. In other cases of disease, however, there is a slighter degree of wasting of the muscles; there is no loss of irritability in the nerves; the irritability may be slightly increased or lessened, but is changed in the same way to each form of electricity. In this condition reflex action is not lost, but, on the contrary, is often increased. The change is apparently the result of a peculiar alteration in the nutrition of the nerve-cells. It is usually the effect of an *irritative* degeneration of the pyramidal fibres, which influences in a peculiar manner the nutrition of the motor cells.

The changes in the nutrition of the skin, if slight and chronic, resemble those produced by disease of the nerves (see p. 79), and are probably produced through the agency of the posterior roots. Perforating ulcer and the trophic disease of joint known as "Charcot's joint" are met with especially in tabes. They also occur in syringomyelia. Sometimes, however, these changes are most acute. Very slight pressure, continued for a few hours, causes vesication, and even sloughing of the skin. Sometimes bullæ form where there has been no pressure. Occasionally there is inflammation of the joints. Such intense changes are only met with when the morbid process in the cord is irritative in its character, especially in hæmorrhage, and in spreading or disseminated myelitis. The points on which sloughing occurs with greatest readiness are the heel, over the malleoli, the trochanters, and the sacrum. The position of the bedsores in paraplegia, usually over the sacrum, is different from that in hemiplegia, generally over the gluteal region on the paralysed side. The tendency to trophic disturbance probably favours the occurrence of cystitis from retention of urine, and the readiness with which the inflammation invades the kidneys. I have seen a low form of suppurating subperitoneal pelvic cellulitis, in a case of disseminated myelitis with cutaneous sloughs, in which no other cause for the cellulitis could be found.

Vaso-motor disturbance is common in slight degree, irrespective of the seat of the disease, but, like the trophic changes, which probably depend in some degree upon it, it is always most intense in irritative lesions of the cord. The slighter and common degree consists merely in increased warmth of the limbs, in which the vessels dilate too readily; while, at a later period, the limbs are colder, paler, and often livid at the extremities. In rare cases intense flushing of the skin has been observed, or copious and long-continued perspiration. When the disease is in the lower cervical region, flushing and perspiration may occur on the face and head.

Disease of the cervical cord or nerves is sometimes attended by a change in the pupil, on one side only. It is due to the fact that the dilator is innervated from the cervical sympathetic through the last cervical nerves. There may be contraction, from paralysis of these radiating fibres, or there may be persistent dilatation from their irritation. There may also be narrowing or enlargement of the palpebral fissure from the same cause. The cause of the double myosis, so common in some degenerative diseases of the cord, is uncertain; it is associated with loss of the light-reflex, and also of the reflex dilatation produced by stimulating the skin of the neck.

The frequency of the heart's action is often permanently increased in locomotor ataxy, but we do not know by what mechanism. In disease of the cervical cord (perhaps also in that of the cervical nerve-roots) the heart may be slowed to forty, thirty, or twenty beats per minute, usually only for a time. This is frequently met with in fracture dislocation in this region. The effect may perhaps be due to a loss of the influence of the nerves which, when stimulated, quicken the heart. In acute disease of the upper dorsal region the heart's action may be persistently frequent.

Visceral Disturbance.—Various disturbances in the *alimentary canal* occur in disease of the spinal cord, and are no doubt due to the fact that the sympathetic nerves immediately controlling the canal are under the influence of the cord, and may be excited to abnormal action by its irritation, or may underact when its influence is lessened. Difficulty of deglutition is observed in some cases of acute disease of the upper cervical region. Vomiting occurs also in disease of the same part, especially in fracture of the cervical vertebræ. Constipation is extremely common in disease of the cord above the lumbar enlargement. The "gastric and laryngeal crises" of tabes are especially frequent in cases in which the disease is situated high up in the cord. When rectum and bladder are affected, the disease must be lower.

Paralysis of the Sphincters.—Incontinence of urine and fæces may be due to disease of the centres that control the action of the sphincter ani and the muscular mechanism of the bladder respectively, or it may be due merely to disease of the path (probably the pyramidal tract) through which voluntary control is exerted. In the latter case the sphincter ani acts in an automatic reflex manner, as already explained. If the finger is introduced within it, the initial relaxation is felt to be succeeded by a firm tonic contraction. This proves that the anal centre in the lumbar enlargement, and the nerves proceeding from it, are intact. If this centre is destroyed, or the nerves interrupted, no tonic contraction can be felt; there is complete and persistent relaxation. I have more than once observed a dorsal transverse myelitis arrest the voluntary influence and leave the sphincter in its condition of simple reflex action, and, a week or ten days later, the extension of the myelitis into the lumbar region, to be attended by (among other signs) perfect inaction of the sphincter. A rectal examination will then often give important information regarding the extent of the disease downwards.

The sphincter of the bladder is not accessible to such direct examination, and the mechanism seems to be more complex, but we may distinguish conditions corresponding to those of the rectum. If the lumbar centre is destroyed the sphincter is permanently relaxed; urine dribbles out of the bladder as fast as it enters it; there is simple incontinence. If there is an interruption of the voluntary path above the centre, the sphincter may act automatically: when a certain amount of urine has collected in the bladder it excites the muscular fibres in the wall to contract; the sphincter relaxes, and the urine escapes,—there is intermittent incontinence. In this condition the mechanism is excited to action by any pressure on the bladder; a movement, or the act of coughing, will make the urine come away. But the muscle of the wall of the bladder is indirectly under the influence of the will; when the sphincter is voluntarily relaxed the detrusor fibres in the wall contract. Loss of voluntary power seems to lead to weakness in the wall of the bladder, so that the bladder is never perfectly emptied, and ultimately the detrusor cannot contract to expel the urine, even when the sphincter is relaxed. Hence retention of urine occurs. When a certain degree of distension of the bladder is attained, the pressure becomes sufficient to force the urine out whenever the sphincter relaxes, and such relaxation is continually occurring under the high pressure that is repeatedly renewed in consequence of the flow from the kidneys. Hence incontinence results,—"overflow incontinence" (see p. 247). The retention often develops very gradually and insidiously when the voluntary power is only lessened; the residual urine is at first small in amount, but gradually increases as the detrusor fails to contract adequately against the resistance. It is important, if the urine comes away involuntarily, to ascertain which form of incontinence exists; since overflow incontinence, and the retention it indicates, have much graver consequences than simple incontinence. The permanent distension of the bladder constitutes a permanent hindrance to the flow of urine from the kidneys, and serious kidney disease may result. This is a not uncommon cause of death in spinal disease. Moreover, whenever the bladder is habitually emptied imperfectly, decomposition is apt to occur in the residual urine. The decomposition is often aided by the lessened acidity, or even actual alkalinity, of the urine. This change in the composition of the urine excites inflammation of the bladder; pus is formed by the mucous membrane, and this, in its turn, increases the decomposition of the urine. The damage to the kidneys, by the hindrance to the flow of urine from them, may be increased by inflammation ascending the ureters from the bladder.

Pyrexia.—The initial elevation of temperature in some spinal lesions may be due to them directly, but sometimes it is due to a blood-state of which the cord disease is a result. This is probably the case in polio-myelitis. The temperature is sometimes raised in disease of the cord apart from the influence of morbid process. Disease of the upper cervical cord, like that of the medulla, may cause hyperpyrexia, and this may also result from acute central lesions that occupy a considerable vertical extent, such as a central hæmorrhage, although by what mechanism these act we do not know. Slight variable elevation of temperature sometimes occurs in disease of the dorsal cord, and is probably due, in some way, to the disturbance of its functions rather than to any influence of the morbid process. For instance, in one case of hæmorrhagic myelitis with damage from the third dorsal to the second lumbar segments, every application of electricity to the legs was followed by slight pyrexia.

Convulsions.—Lastly, convulsive attacks have been known to attend the onset of acute lesions in various parts of the cord, in adults as well as in children. They are most frequent in disease of the cervical cord, but I have known a convulsion to occur in an adult at the onset of myelitis in the lower dorsal region. The convulsions are general. Their mechanism is unknown, and in some cases, especially of infantile polio-myelitis, they may, like the initial pyrexia, be the result of general disturbance of the system by an acute morbid blood-state.

Optic neuritis has been met with, especially in connection with disease in the cervical region of the cord, and in cases in which careful examination of the brain post mortem failed to reveal any cerebral disease.

COMBINATION OF SYMPTOMS.—Certain symptoms are frequently combined, and their combination has a definite localising significance. Loss of motor power occurs in two different associations, according to the position of its cause in the motor path. It may be combined with muscular wasting, loss of electric irritability, and loss of all reflex action. In this case the disease is in the anterior cornua or nerve-roots, the lower segment of the motor path. (But the same symptoms are produced by disease of the nerves themselves—in multiple neuritis.) On the other hand, there may be no wasting, no change in irritability, and, instead of a loss of reflex action, the myotatic irritability is increased. The interruption of the motor path is then in the upper segment, usually in the pyramidal tracts. It may be a focal lesion of the cord, or a primary degeneration of the tracts. The distinction is that, in the latter case, there is nothing more than the muscular weakness and increased myotatic irritability. In the former case there are, or have been, indications that the lesion has extended beyond the purely motor elements.

The symptoms caused by disease of the several elements of the cord have now been described, but it is necessary to consider further the combination of symptoms that results from a total transverse lesion of the cord, and from a unilateral lesion.

A *total transverse lesion*, however limited in vertical extent, separates from the brain all parts below it, and hence, so far as will and feeling are concerned, it produces the same effect as if the whole of the cord below the lesion were destroyed. Compression of the cord in the middle of the cervical enlargement, for instance, abolishes motion and sensation in all parts below the distribution of the cervical plexus. A transverse lesion at the second dorsal nerve causes motor and sensory palsy of the trunk and legs, leaving the arms unaffected. But the same loss, motor and sensory, results from disease which damages the whole cord up to the level indicated. Hence the extent of the motor and sensory paralysis indicates only the upward limit of the lesion; how far it extends downwards is shown by the interference with the central and especially the reflex functions of the cord. The upward level is indicated not only by the loss of conduction, but also by the position of the girdle-pain and radiating pains, or zone of hyperæsthesia, which are due to the irritation of the lowest sensory nerves in the upper portion.

It is desirable to know the symptoms of impaired conduction that occur when a transverse lesion is at different levels in the spinal cord. They may readily be ascertained by an examination of the table and figures given at pp. 250—252. There is, however, some uncertainty as to the effect on sensation in the limbs produced by disease at different parts of the lumbar and cervical enlargements, since, as we have seen, the exact representation of sensation in the cord is still uncertain.

The upper limit of the lesion is shown by the upward extent of the motor and sensory loss. The lowest nerves supply the anus and perinæum. Those that supply the skin and muscles of the leg and foot arise from the fourth lumbar to the second sacral segments, and are damaged by a lesion involving the lower part of the lumbar enlargement. In the middle of the lumbar enlargement the nerves arise which enter the lumbo-sacral cord, and these are probably destined for the flexors of the knee, and for the hip muscles supplied by the sacral plexus, the glutei, quadratus, and gemelli, and for the skin of the lower part of the gluteal region. These parts then will be paralysed by disease in the middle of the lumbar enlargement, while the muscles and skin in front of the thigh are unaffected. The latter suffer when the disease affects the upper part of the lumbar enlargement, the origin of the anterior crural (rectus, &c.), and obturator (adductors). The skin on the upper and outer parts of the thigh loses sensibility, with the part adjacent to the scrotum, and in the groin, only when the disease damages the highest part of the lumbar enlargement, from which the second and third lumbar nerves arise, and then the flexors of the hip become paralysed. In proportion as the disease is higher in the dorsal region we have the symptoms ascending higher up the trunk, and marking accurately the height of the lesion by the loss of cutaneous sensibility, and by the impairment, first of the abdominal muscles, and then of the intercostal muscles. The umbilicus corresponds to the tenth dorsal nerves, and the ensiform area to the sixth and seventh. When the disease reaches the lowest part of the cervical enlargement (the first dorsal nerves) we have the first symptoms in the upper extremity; but these are not, as might be expected, in the muscles moving the shoulder-joint, but in the hand. The first numbness is complained of in the little finger, and the first weakness is in the intrinsic muscles of the hand. Ascending higher, the symptoms pass up the arm with some uniformity, and without respect to nerve distribution. When the middle of the cervical enlargement is reached (the fifth, sixth, and seventh cervical) the shoulder muscles and the serratus magnus become paralysed, and there is general loss of power and sensation. (For details see table, p. 252, and also pp. 250 and 251.) Above the level of the sixth pair the trapezius and sterno-mastoid become somewhat weakened, for the fibres of the spinal accessory which supply them undoubtedly arise in part from this region of the cord. At the fourth and fifth cervical the lower part of the neck becomes anæsthetic, and the diaphragm ceases to act. Here our localisation might cease, for total transverse lesions at this spot necessarily cause death. For a little time the sterno-mastoids and scaleni can still get some air into the chest, rarely in sufficient amount to maintain life for more than a few days. But limited lesions may occur higher up, and then we have complete powerlessness of the muscles moving the head, upper part of trapezius and sterno-mastoid, and other muscles attached to the occipital bone, and interference with sensation in the neck and parts of the head, which are not supplied by the fifth nerve.

The extent downwards of the lesion, its vertical extent, is thus not indicated by the impairment of the conducting functions, by the motor or sensory paralysis; to ascertain it we have to examine the functions of the cord as a central organ, and to ascertain how far they are impaired in the paralysed region—to examine especially muscular nutrition and reflex action. The state of muscular nutrition and irritability indicates how far the anterior cornua are injured. The relation of the several groups of muscles to the cord is shown in the first column of the table. The integrity of reflex action indicates the integrity of the reflex loops, and the study of the superficial reflexes of the trunk is especially instructive in this respect. The series of reflexes, and the relation of each to the cord, are shown in the second column of the table; the muscle-reflex contractions are printed in italics in the position which corresponds to the centres and nerve-roots which are essential for their production. Excess of superficial reflex action indicates loss of the cerebral control exerted on the reflex centres, and considerable excess of the muscle-reflexes implies impaired function of the lowest part of the pyramidal tracts, and generally shows the existence of a descending degeneration in the lateral columns. Reflex action in the legs may be abolished for a few hours by acute disease or injury above the lumbar enlargement, in consequence of inhibitory shock, but soon returns if the lumbar centres are intact. As already explained (p. 264), if there is no return of reflex action or myotatic irritability, their absence shows that the lumbar region is involved in the primary lesion; secondary extension to this is indicated by loss of reflex action distinctly subsequent to the onset. If such loss occurs without wasting and loss of faradic irritability in the muscles, it shows that the disease in the lumbar region is limited to the posterior columns or cornua.*

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* A few cases have been met with in which a transverse lesion of the dorsal cord, especially a transverse concussion-myelitis, without post-mortem signs of lumbar inflammation, has caused a persistent loss of reflex action in the legs. In

The fact of chief importance is that dorsal cord disease, the most common form, causes no persistent loss of reflex action in the legs, and that for such loss special causes must be sought.

Unilateral lesions interrupt the motor path to the muscles on the same side as the lesion, causing one-sided palsy, termed "spinal hemiplegia" when the disease is so high as to affect both arm and leg, "hemiparaplegia" when it is lower, and affects one leg only. There is often some loss of power on the opposite side, which may be due either to slighter damage to the other side of the cord (since few lesions are strictly unilateral) or to damage to non-decussating fibres. Conversely, the paralysis of the leg may be incomplete when that of the arm is complete, owing to the escape of the fibres for the leg which cross lower down the cord. Sensation is affected on the opposite side, but not quite up to the level of the lesion, because the decussation of the sensory tract is not immediate, but occurs somewhat above the entrance of the nerves. The upper level may vary for different forms of sensibility, in consequence probably of the level of crossing (in relation to entrance) being different for the several paths. A lesion in one side of the lumbar enlargement often affects sensation on the same side as motion, because it damages the sensory path before it has crossed. In all cases of crossed motor and sensory paralysis the sensibility of the muscles differs from the other forms of sensibility; and if it is affected on one side, this is the side of the motor palsy, and not of the cutaneous anæsthesia.*

The crossed affection of cutaneous sensibility may involve all forms of sensation or only some of them. Sensibility to pain is almost in-

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these cases, however, there has been not only loss of the muscle-reflex action, but also of the skin reflexes, a clear indication of an exceptional condition. Moreover there has also in other cases been loss of faradic irritability and rapid muscular wasting,—in short, there have been all the indications of a lumbar myelitis. The most probable explanation of these cases is that the descending degeneration of the pyramidal tract has been more than usually irritative in nature, so as practically to amount to a parenchymatous inflammation, and that this has invaded the motor structures as a nutritional change, sufficient to abolish their function, without, however, destroying their form. In one such case it was noted that the lumbar nerve elements were extremely granular in aspect. For a different explanation of these cases see Bastian, 'Med.-Chir. Trans.,' 1890; Bruns, 'Neurol. Centralb.,' 1895, and 'Arch. f. Psych.,' xxv; and Egger, 'Arch. f. Psychiat.,' xxvii; see also note, p. 264.

* This was first pointed out by Brown-Séquard in his important study of these cases. Scepticism has been expressed as to the fact, based on a denial that the test commonly employed, recognition of posture, is significant. Experiments on animals, even monkeys, are inconclusive, but the evidence from cases in man is very strong, not only of the occurrence of the loss, but of the significance of the test. In some cases, moreover, the muscles were insensitive to pressure. One recorded instance is the following:—A stab in the dorsal region caused loss of cutaneous sensibility in the right leg and motor palsy in the left. On the left side, on which cutaneous sensibility was normal, the sense of posture was absolutely lost, while it was present on the right side, on which cutaneous sensibility was impaired. The patient often thought the left leg was flexed when it was extended. Ultimately this leg presented distinct ataxy. (Gilbert, 'Arch. de Névrologie,' 1882, p. 275.)

variably impaired. The temperature sense is usually affected with that for pain; in only two of twenty recorded cases (in which the affection of sensibility was carefully noted) was the sense of temperature normal, and that of pain impaired, and in neither of these cases was the sensibility to pain actually lost. On the other hand, in one third of the cases tactile sensibility was unaffected, and in about one tenth of the cases it was impaired on both sides.

Cutaneous sensibility is sometimes impaired in a zone at the level of the lesion, and on the same side, in consequence of the damage to the nerve-roots entering the cord, and above this there may be a narrow band of hyperæsthesia from irritation of the roots at the upper part of the lesion. On the side of the lesion, below the anæsthetic zone, there is, in most cases, a remarkable hyperæsthesia, corresponding in distribution with the anæsthesia on the opposite side. Painful impressions are felt most acutely, and even a touch on the skin, or warm or cold bodies, produce pain. The cause of this hyperæsthesia is obscure. Both it and the opposite loss of sensibility may last for twenty years, and it cannot be ascribed therefore to any irritation by the morbid process. It is perhaps due to an altered action of the cerebral centres on the opposite side of the brain. There is much evidence of an intimate connection between the sensory centres on the two sides, and it is conceivable that the altered functional state of the centre on the side of the lesion, to which impressions cease to come, may induce in the opposite hemisphere a condition expressed as hyperæsthesia. The condition may be thought of in relation to the peculiar phenomena of transfer in hysterical hemianæsthesia.

Reflex action is increased in all its forms on the side of the lesion, but the increase only occurs after some days. At first it is lessened or even abolished, no doubt from the inhibitory influence of the irritation of the morbid process.

The side below the lesion is at first, for some days or even weeks, warmer than the other, the difference being usually about a degree Fahrenheit. In the course of time this difference passes away, and the side may even be colder than the other.

These symptoms may be shown in the form of a table:

Zone of cut. hyperæsthesia. ——— anæsthesia.	LESION.	
Motor palsy. Hyperæsthesia of skin. Muscular sense impaired. Reflex action first lessened, then increased. Temperature raised.	} {	Muscular power normal. Loss of sensibility of skin. Muscular sense normal. Reflex action normal. Temperature same as that above lesion.

The vertical distribution of the symptoms varies according to the vertical position of the lesion. Their relations are best marked when the lesion is in the dorsal region of the spinal cord. When it is in the cervical or lumbar enlargement the upper limit is less distinct because of the complex representation of skin and muscle in the nerve-roots.

GENERAL PATHOLOGY AND ETIOLOGY: INDICATIONS OF THE NATURE OF THE LESION: PATHOLOGICAL DIAGNOSIS.

The kinds of disease to which the spinal cord is liable are not specifically numerous, but those which are common present several varieties. The processes are congestion and anæmia, hæmorrhage, inflammation, degeneration, and growths. Hæmorrhage and growths are, on the whole, rare. Congestion plays an uncertain part in the production of symptoms; its influence has been certainly overrated by some writers, and perhaps under-estimated by others. Anæmia is now known to be sometimes associated with certain definite changes in the spinal cord, and it is possible that it may occasionally give rise to symptoms without producing gross disease. The most frequent lesions are inflammation and degeneration. These two processes, distinct in their typical forms, blend in their intermediate varieties. Inflammation varies much in character and course. Its effects are in some cases produced with great rapidity, in other cases with extreme slowness. It probably commences in most instances outside the nerve-elements, which are secondarily involved. Perhaps some acute processes in the nerve-elements themselves are most accurately regarded as a parenchymatous inflammation, analogous to that which we know occurs in the fibres of the peripheral nerves. Degeneration consists of two processes, a wasting of the nerve-structures, cells and fibres, and an overgrowth of the connective-tissue elements. The latter leads to the condition termed sclerosis. The name has been given to it, apparently, because the process is one that, in other organs, increases their consistence. In the cord it has not often this effect. The newly formed connective tissue is rarely of greater firmness, and is often less firm, than the nerve-elements it replaces. In sclerosis, however, we must distinguish two types. In one the process is limited to structures that have the same function; the process begins as a degeneration of the nerve-elements, and the overgrowth of the connective tissue is a consequence of their wasting—the affection is essentially "parenchymatous." The so-called "secondary degenerations" are of this character; but similar changes are often pri
and affect the structures that have the same function, often thr
considerable extent of the cord. Hence they are term "
diseases." In the other type the morbid changes are not

according to function. They are apparently random in incidence, and involve adjacent structures in consequence of contiguity. These begin in the connective tissue and not in the nerve-elements, which suffer secondarily. They are essentially interstitial processes. Insular sclerosis is an example of this type.

The position of the islets of sclerosis has no relation to the function of the parts, and may involve parts of adjacent structures that have no common function. Areas of diffuse sclerosis are also met with which have not the sharp limitation of the insular form, and are regarded by some as of the nature of chronic sclerotic inflammation. If such a lesion is so situated as to involve fibres that undergo secondary degeneration, this necessarily results when the damage to the fibres is sufficiently great. We have then a combination of the random and systemic forms of sclerosis, but one that has no real pathological significance.

There are, however, other relations between the two types that deserve attention. These will be considered in connection with the special morbid processes that present them, but a brief mention of them here may make some facts of other diseases more intelligible. The process of secondary degeneration and secondary sclerosis varies in its character, presenting in some cases a greater amount of vascular disturbance than in others. It seems to partake of the character of the lesion causing it, as we have seen (p. 73) the process of secondary degeneration in the nerves may do. The same difference is seen in the primary degenerations, such as occur in tabes. This difference is analogous to that which, as already described, occurs in some varieties of multiple neuritis. We can indeed see the difference in the eye: tabetic optic nerve atrophy, in most cases, presents no sign of inflammation, while in others the early stage may be accompanied by distinct slight neuritis, to be seen with the ophthalmoscope. Secondly, the chronic inflammations, with the diffuse limitation, extending to adjacent structures irrespective of function, have yet sometimes a marked tendency to be localised in structures of definite function. Thus there is sometimes diffuse sclerosis of the lateral pyramidal tracts and posterior median columns, not sharply limited to these, and yet with a correspondence and symmetry not to be overlooked. Thus the two processes tend to meet; the systemic degeneration or sclerosis to be diffuse; the diffuse inflammation or sclerosis to be systemic; and it is sometimes not easy to say, from mere microscopic examination, to which class a lesion belongs. Lastly, there is a curious fact, which has been strangely overlooked although attention was first called to it by Charcot—that inflammation may extend from a primary lesion along a tract that undergoes secondary degeneration, but in the opposite direction to that of the degeneration. It is very common for the inflammation at a given place in the cord, especially that due to compression, to extend farthest along certain tracts, irrespective of their secondary degeneration, and in some cases this extension occurs through a considerable extent of the cord. An ascending inflammation limited to the pyramidal tracts may, for instance, be traced through several segments. The fact suggests that the neuroglial elements may share to some extent a difference that obtains between the fibres of different tracts, and it may easily lead, and probably has led, to erroneous conclusions.

Softening of the spinal cord is very common. Whenever the nerve-fibres are broken up into disconnected globules of myelin, separated by serum in a sort of emulsion, the consistence of the part is necessarily lessened. Acute inflammation always causes such a breaking up of the nerve-fibres, and hence inflammation always causes softening as its first effect. The separate particles are augmented by leucocytes, which escape abundantly into the tissue. Ultimately connective-tissue elements are formed, which increase the consistence, it may be up to, and even beyond, the normal degree. In a very chronic inflammation the formation of new tissue may proceed *pari passu* with the destructive process, and there may be at no time much diminution of consistence. It is in these cases that it is difficult to draw the line between inflammation and degeneration.

Does such softening of the cord occur apart from inflammation? The question is not easy to answer. In the brain necrotic softening is very common as a consequence of arterial obstruction—is indeed the common form of softening. But in the spinal cord we have no such distinct evidence of the occurrence of necrotic softening. If embolism occurs, it is excessively rare. The course of the vessels does not favour the passage of a plug into them, while the vertical connection in the anterior spinal and anastomotic arteries (see p. 227) will prevent damage from obstruction in the central system unless this is situated in the terminal vessels of the grey matter. Arterial thrombosis, due to atheroma of the walls of the vessels, such as is so common in the brain, probably does not occur in the cord. The arteries are smaller than those in which atheroma is met with in the brain, and the lowness of the blood-pressure within them involves the absence of the chief cause of atheroma. It is likely that spontaneous thrombosis occurring in vessels affected with syphilitic endarteritis sometimes sets up the changes that are now regarded as those of primary inflammation; and such a lesion has actually been met with, but we have no evidence of its frequency.*

Besides the morbid processes to which the cord itself is liable, it suffers also in consequence of disease outside it. It may be compressed by growths springing from the membranes or bones, and by products of inflammation within the spinal canal. Compression not only causes degeneration of the nerve-elements, but usually excites actual inflammation. This "compression-myelitis" may attain a degree and an acuteness out of all proportion to the causal compression. The membranes may be the seat of hæmorrhage which compresses the cord, or inflammation which irritates it, even when the organ is not invaded in appreciable degree.

The series of recognised lesions of the spinal cord, obvious or microscopic, does not by any means exhaust the list of its morbid states. Changes may occur in the nutrition of its elements interfering with their function, which are, and are likely to remain, altogether beyond our means of detection. Such morbid states are often called "functional diseases," but when prolonged they are probably more accurately conceived as nutritional diseases (see p. 1). Derangements of function that we can regard as purely such are few and rare. But disturbances of function may be due to organic disease in some other part of the nervous system, and they may also be the result of toxic agents.

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* Williamson, 'Lancet,' 1894.

Lastly, functional action in all organs is attended with increased blood-supply; and when excessive, the vascular disturbance may go on to a condition indistinguishable from inflammation, with escape of leucocytes, &c. The prolonged experimental stimulation of sensory nerves has actually produced myelitis in the related portion of the cord.

In connection with the general pathology of diseases of the spinal cord, it is important to note certain general facts of their causation:—(1) *Neuropathic disposition.*—An inherited tendency to disease of the nerve-elements, manifested by such affections as epilepsy and insanity. This cause is chiefly influential in producing the structural and nutritional diseases, beginning in the nerve-elements, and especially, among structural diseases, the "system-degenerations," as they are called. (2) *Prolonged mental distress* sometimes sets up degenerative changes in some elements of the cord, especially in persons who are predisposed by (1). (3) *Injury.*—A severe concussion of the cord may cause (*a*) instant grave damage, usually hæmorrhage. Or (*b*) it may cause no immediate effect, but symptoms may come on at the end of a few days and progress slowly or rapidly. Such symptoms are usually due to inflammation, secondary to minute spots of injury, too small to cause direct symptoms. Lastly, (*c*) the concussion sometimes seems to pervert the process of nutrition in the nerve-elements. Slow symptoms of impaired function may result, and these may progress until, after months or years, there is actual structural disease. Similar effects, especially the graver varieties, are met with as a result of lessened atmospheric pressure in divers and workers in caissons. (4) *Exposure to cold.*—Wet cold is especially effective. The most frequent effect of a severe exposure is acute inflammation, but habitual exposure may produce chronic inflammation, or degenerative disease. The exposure that is effective may be general, or chiefly of the feet and legs, occasionally of the back. (5) The spinal cord appears also to be the seat of primary infection as in epidemic cerebro-spinal meningitis, tetanus, and possibly acute anterior polio-myelitis. (6) *Toxic agents* that can act on the cord are numerous, and are seen in alcoholism and lathyrism. (7) *Other diseases* may also induce morbid changes in the spinal cord. The primary malady may be an acute specific disease, or some more obscure blood-state, the precise nature and relations of which are still undecided. It is probable that certain blood-states due to an organised virus excite some forms of inflammation of the cord and of its membranes, just as it may cause inflammation of the peripheral nerves. Septicæmia may excite meningitis, or even abscess of the cord itself. Tubercle is common on the membranes, but seldom produces inflammation. Diphtheria may cause acute changes in the nerve-cells and nerve-roots. Gonorrhœa * has produced myelitis, and such diseases as variola, typhus, &c., have been followed by cord inflammation. Syphilis is a very frequent cause of disease, and may act in several ways. The demonstrated mechanisms are (*a*) by a syphilitic growth compressing or invading the cord, (*b*) by changes in the cord resulting from thrombosis occurring in diseased vessels, and (*c*) by chronic syphilitic meningitis, damaging the cord and the nerve-roots. But (*d*) acute and chronic inflammations of the cord are often met with in syphilitic subjects, and have been thought to be, in many instances, of syphilitic origin. It is doubtful whether they present any syphilitic characters, *i. e.* any histological features by which they differ from non-syphilitic inflammations. The evidence of dependence on syphilis is stronger in the case of chronic and subacute disseminated inflammation than in the case of acute myelitis. It may be, however, that inflammations due to other causes, *e. g.* exposure, run a more acute course when they affect syphilitic subjects. The part thus played by syphilis in lessening the resisting power of the organism may be shared by other factors, such as malnutrition and excesses of various kinds. Lastly, (*e*) certain degenerative diseases of the cord are very commonly preceded by syphilis; one of them, locomotor ataxy, so frequently that a causal relation between the two can scarcely be doubted. But these degenerative diseases are certainly not syphilitic in nature; they differ in no respect from the similar morbid processes that occur in individuals who have not had syphilis, and are probably the consequence of some toxin left behind by the morbid agency to which the constitutional malady is due. (8) Excessive muscular exertion has been held to play a part in the generation of chronic disease of the cord. This view has of late derived some support from experiments upon animals.† (9) Pathological changes in the cord are known to result from the amputation of limbs. These have, however, no clinical significance.

The outline just given of the general pathology and etiology of diseases of the spinal cord will enable us to consider the last element in diagnosis, the nature of the lesion. The seat of the disease is indicated by the combination of symptoms; its nature can only be determined by considering, separately and together, several other points:—(1) The way in which the symptoms came on. (2) The causes that can be traced, taken in conjunction with the known effects of those causes. (3) The seat of the disease, taken in conjunction with the known liability of certain structures to certain lesions. The most important of these elements is the mode of onset, and the other indications should only be used in strict subordination to this.

* Gowers, 'Clinical Lectures on Nervous Diseases,' p. 119; also Leyden and Goldscheider, 'Die Erkrankungen des Rückenmarkes,' p. 369.

† Independently by O. Rosenbach and Edinger (see Edinger and Helbing, 'Verhandlungen des 16 Cong. f. inner. Med.,' Wiesbaden, 1898).

The time occupied by the onset of the disease is thus the first element in the pathological diagnosis. By "time of onset" is meant the period that elapses between the actual commencement of the symptoms and their attainment of a considerable degree of intensity. We may divide the chief modes of onset into five classes, and classify the most common lesions in relation to them, in the following table:

DISEASE.	ONSET.	DISEASE.
	Sudden (few minutes)	Vascular lesions.
	Acute (few hours or days)	Inflammation.
Pressure and growths	*Subacute* (one to six weeks)	
	Subchronic (six weeks to six months)	Degeneration.
	Chronic (more than six months)	

A lesion of sudden occurrence, the symptoms developing in the course of a few minutes, is almost always vascular, commonly hæmorrhage, sometimes perhaps vascular obstruction. But a vascular lesion may occupy a somewhat longer time in development—a few hours or days. In acute and subacute inflammation the symptoms come on in the course of a few hours, a few days, or a few weeks. Subacute and chronic inflammation occupies from a few weeks to a few months. Degeneration, in which there is no adequate evidence of any inflammatory process, occupies many months, or it may be years. The symptoms produced by tumours which invade or compress, and by simple pressure (traumatic causes excluded), are never sudden or very acute, and rarely very chronic; the time occupied by the development of the symptoms varying, according to the nature of the cause, from a fortnight to six months.

It is necessary to consider, however, not merely the whole time occupied by the development of the disease, but also the uniformity of its course. Two or more morbid processes may concur. An initial myelitis, for instance, may lead to a secondary degeneration; and on the other hand, in degenerated tissues, sudden vascular lesions occasionally occur. Pressure produces local myelitis, which may be independent of the pressure in its development, and have an acute or subacute onset. The whole course of the disease must be ascertained before an inference is drawn, and the possibility of a double process must always be kept in view.

The onset and course of the symptoms thus sometimes enable us to decide at once that a lesion is of a given character, as that one which occurs instantly is vascular, or that one which takes years for its development is degenerative. More frequently they enable us to exclude certain morbid processes, and to restrict the possible lesion to two or three forms. For instance, a lesion which comes on in the course of a few hours must be either vascular or inflammatory. Between these we have to decide by attention to other indications.

In actual diagnosis it is convenient to consider next the indication afforded by the position and distribution of the disease. We consider what diseases occur in this situation, and then which of them have the mode of onset that has been ascertained. This indication involves a knowledge of the various diseases and their seat. The most important consideration is that a wide range of symptoms of uniform character indicates the affection of a definite system of structure, and in most instances a disease commencing in the nerve-elements, and if the onset be chronic we may feel sure that it is a degeneration. On the other hand, the involvement of many functions suggests a random process, such as inflammation or pressure. But this indication is always to be subordinated to the mode of onset. Thus the limitation to a single structure does not exclude inflammation: this may affect, for instance, the anterior grey matter only, and cause corresponding symptoms.

The symptoms may indicate a morbid process limited to one half of the cord, but this does not materially modify the diagnostic method. Almost any process may, in rare cases, be thus limited. System degenerations and acute inflammations are least frequently unilateral, and they never reach a considerable degree of intensity on one side without some affection of the other side. On the other hand, tumours and foci of chronic myelitis are often one-sided, and still more often affect one half of the cord first and then the other.

Indication of disease outside the cord, irritation of certain nerve-roots, causing severe local pain, often precedes the symptoms of compression, and is an important aid to diagnosis. It shows the existence of a morbid process outside the cord before the cord is involved. But we cannot use even this indication except in dependence on the mode of onset. A disease, as a growth outside the cord, may, as we have seen, not only compress the cord, and cause slow loss of power; it may excite inflammation and cause rapid palsy.

The last element in the pathological diagnosis is the detection of any influence which can be regarded as the cause of the disease in the spinal cord, or any associated condition which may indicate an active morbid process. We have seen that the mode of onset may help us to limit the disease to certain possible forms of lesion; the distribution of the affection may render it probable that it is one or other of these forms; and the detection of a cause and the knowledge of the lesion which that cause produces may help us to carry the diagnosis still further. The most important general causes of disease of the cord, and the processes to which they chiefly give rise, have been already mentioned. The causal element in diagnosis is chiefly an application of those facts.

The morbid process outside the spinal cord that most closely simulates its disease is, unquestionably, parenchymatous multiple neuritis.

The diagnosis depends on a thorough knowledge of the varied symptoms of the latter, since its elements vary according to the different manifestations of the disease of the nerves. For these, and for any general diagnostic principles, the reader is referred to the account of that disease.

The only sure ground for diagnosis is a thorough knowledge of the various morbid processes and their symptoms; and the only safe plan is to work by these, from symptom to seat and onset to nature, treating every case as a problem to be worked out to a definite diagnosis, and only then comparing the result with the types of disease. To one of these the case may or may not conform; if it does not, the comparison with types as a means of diagnosis will only leave the observer stranded and powerless.

The distinction of functional and nutritional disease from organic lesions may conveniently be postponed until the symptoms of the former are specially described.

SPECIAL DISEASES OF THE SPINAL CORD.

DISEASES OF THE VERTEBRAL COLUMN.

Diseases of the bones of the spine fall for the most part within the province of surgery. But there are few of these diseases that do not, among their most frequent effects, interfere with the functions of the spinal cord. Hence an account of the diseases of the cord would be incomplete without some mention of the morbid states that begin in its bony case.

INJURIES OF THE SPINE.

Injuries to the spinal column may consist of punctured wounds (which need not be considered here), concussion (the effects of which on the cord will be considered later), and fracture or dislocation of the vertebræ. It may, however, be useful briefly to describe the more salient features of the last two conditions, their relation to the damage the cord sustains, and the symptoms thus produced.

Dislocation.

Simple dislocation occurs in the cervical region, most frequently at the fifth and sixth vertebræ. It may take place gradually or suddenly: gradual displacement is always secondary to disease of the bones;

sudden displacement may occur in disease or from injury. The damage to the cord is always greatest in traumatic displacement of healthy bones, because the force needed to produce the dislocation is much greater, the displacement is more considerable, and the effect on the canal and the contained cord is greater. Displacement may occur in any direction: it usually involves both vertebral articulations, but, in rare cases, it is oblique, involving one articulation only. The common causes are violent blows or falls on the head, rarely sudden rotation of the head while a weight is carried upon it. The symptoms are a lateral or forward or backward displacement of the head, so that the chin is in contact with the shoulder or the chest, or the occiput with the nape of the neck. There is also irregularity of the vertebral spines, usually readily detected. The cord is damaged in most cases, and the symptoms are those of a total transverse lesion in the situation of the luxation. It may be merely compressed, especially in cases of disease, in which the displacement has occurred with little force. The symptoms of paralysis have been known to pass away, in such a case, on the reduction of the dislocation. More commonly the cord is also bruised, with extravasation of blood, and secondary myelitis occurs later. In such cases, if the patients live, there may be anæsthesia or hyperæsthesia below the lesion, with total paralysis of the limbs, and excess of reflex action. In rare instances the cord has been completely divided. In still rarer instances of slight displacement it has not been injured.

Rupture of the transverse ligament which retains the odontoid process may permit the latter to compress the cord, and thus to cause instant death. This often results from sudden suspension by the head, as in criminal executions. One of the curiosities of surgical literature is a case related by Petit in which a man, playing with a neighbour's child, lifted it up by the head, and caused instant death by rupturing the transverse ligament. The father of the child, entering at the moment, stabbed the man with a knife, the blade of which passed in between the first and second cervical vertebræ, divided the spinal cord, and the man also fell dead. Rupture of the ligament has also resulted from raising a heavy weight with the head. The treatment of dislocation is too purely surgical to be described here.

FRACTURE.

All organic diseases of the bones, weakening them, predispose to fracture. Among these, one is of special medical interest; the vertebræ, especially in the lumbar region, may share the rarefaction and weakening of the osseous tissues occasionally produced in tabes, and then a very slight traumatic influence, a blow or wrench, may cause fracture. The force needed is often so slight as to fail to cause displacement or other symptoms except local pain, and symptoms may only supervene at a somewhat later date. Apart from disease, the accident is most common in adults, the greater elasticity of the vertebral column in children giving to them a comparative immunity. Its cause is a blow or fall on the spine, or sudden forcible flexion. In extremely rare cases a fracture, usually slight, has resulted from severe muscular exertion. This is an important fact, showing that muscular exertion may injure the spine, and may lead to symptoms at a later date. Symptoms of such injury, in slight degree, are not uncommon.

Fracture may occur at any part of the spine, but is most frequent at the fifth or sixth cervical, and at the last dorsal or first lumbar vertebræ. In the dorsal and lumbar regions the bodies are broken in two thirds of the cases, but in the cervical region the arches alone are fractured in one half. Usually there is a displacement of the vertebral column at the seat of fracture. Rarely the bodies may be crushed without displacement. The displacement of the bone involves a narrowing of the canal and, usually, compression of the cord. This may also result when the arches only are driven in. But the cord may be seriously damaged when there is no permanent narrowing of the canal, as in Figs. 89 and 90. The dura mater is rarely torn except by a splinter. Blood is almost always extravasated outside the dura mater, often in considerable quantity, from the rupture of the large veins in this situation. There are usually only small extravasations in the pia mater. The cord is, in most cases, bruised and compressed by the lower fragment (Fig. 91). Sometimes it is flattened, and it may even be divided, all nerve-substance being squeezed out of the pia-matral sheath at the spot. In the case shown in Figs. 89 and 90 the cord appeared to have been split longitudinally at the spot, perhaps by the mere force of the concussion. It is very common to have local myelitis, opposite the fracture, without any permanent narrowing of the canal or compression of the cord; the inflammation seems to be

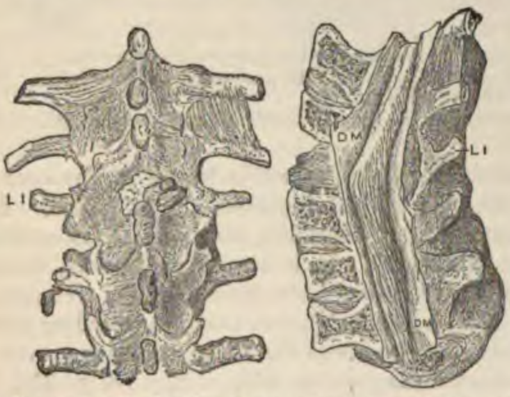

FIG. 89.—Fracture of the first lumbar vertebra. D M. Dura mater. There was no permanent narrowing of the canal, but, nevertheless, the spinal cord was greatly damaged at the spot; see next figure.

the direct result of the concussion. Blood may be extravasated into the bruised part, sometimes in minute spots, sometimes in larger hæmorrhages, and even into the central canal. These changes are usually limited to the spot directly damaged, but secondary myelitis may be set up, and sometimes extends beyond the contused area. It may even extend through the whole length of the cord below the injury, and then its central functions are abolished. In cases of some duration the usual ascending and descending secondary degenerations are also found. It is important to note, moreover, that the secondary degenerations have sometimes the irritative character already described (p. 272, *note*), and that an ascending inflammation may for a short distance above the lesion be limited to a tract that degenerates downwards (see p. 275). Fig. 76, p. 214, represents sections of the cord from the case of fracture figured above. Occasionally the injury leads to secondary caries of the bone, with all its consequences.

FIG. 90.

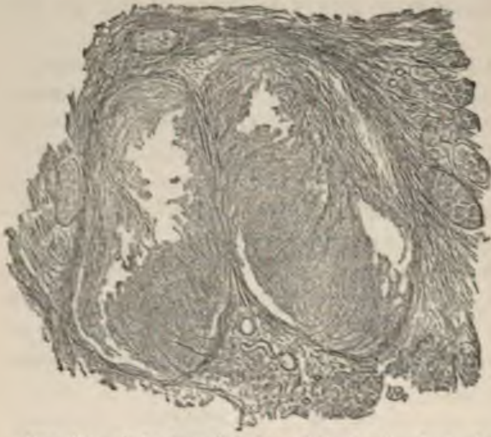

FIG. 91.

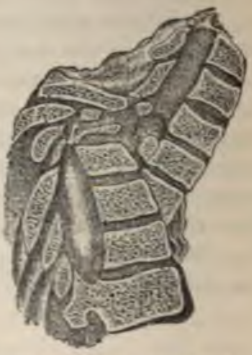

FIG. 90.—Spinal cord damaged by the fracture shown in Fig. 89. The elements of the cord itself are changed beyond the possibility of identification. The ascending degeneration is shown in Fig. 76.

FIG. 91.—Fracture of the body of the fifth dorsal vertebra and of its processes. (After Gurlt.)

SYMPTOMS.—Three classes of symptoms result. (1) The local indications of the injury to the spine. (2) There may be certain nervous symptoms not distinctly due to the damage to the cord. One of these is general shock, which may be so great as to entail transient loss of consciousness. Vomiting occasionally occurs. There is great pain in the position of the fracture, rendered very intense by pressure, and often radiating along the nerves which come from this part, the roots of which are compressed. In rare cases epileptiform convulsions have followed fracture, usually at an interval of some days. (3) Symptoms which result from the damage to the cord, and consist in paralysis of the parts below the injury. Its character depends on the amount of damage. If this is considerable, there is both motor and sensory paralysis up to the level of the lesion, with loss of power over the sphincters. Reflex action is lost at the level of the lesion, and the examination of the trunk-reflexes often gives important information regarding the extent of the damage, when this is in the dorsal region. Below, reflex action is usually diminished or lost, probably because the centres are impaired by descending myelitis. Spasmodic twitchings sometimes occur in the limbs immediately after the injury, and priapism is the rule, especially if the lesion is cervical. The pains are severe in the arms when the fracture is opposite the cervical enlargement, and in the legs when at or below the lumbar enlargement, so as to damage the nerve-roots. In these cases there may be rapid wasting of the muscles, with loss of electric irritability. There is usually at first incontinence, afterwards retention of urine, but the former is persistent if the lumbar centres are damaged. Cystitis, bedsores, &c., may supervene. Ultimately, if the damage is above the lumbar enlargement and the patient lives, there may be increased myotatic irritability in the limbs, progressing to spasm, so that spastic paraplegia results. In many cases in which the knee-jerk is at first lost it may be months, or even years, before it returns.

Special symptoms result when the injury is in certain parts of the spine. Fracture of the *first two cervical vertebræ* causes instant death, unless the displacement is very slight, and even then there is imminent danger of further displacement, with the most serious consequences, on any incautious voluntary movement. With slight displacement persons have been known to live for weeks, and then die from secondary myelitis; they have even recovered altogether. Now and then there is no compression of the cord, although there is distinct displacement, even sufficient to be recognised in the pharynx (Leyden). In such a case death has resulted at a later period from caries. The characteristic symptoms are local pain, increased by all movements (which are rendered almost impossible), displacement, and spinal symptoms. The latter may be slight—merely difficulty in breathing or swallowing—or considerable, and involving the trunk and limbs. Sometimes there is hyperpyrexia. Not more than one case in fifty recovers.

Middle Cervical Vertebræ.—The third, fourth, and fifth vertebræ are most frequently fractured. When the injury to the cord is considerable, death usually occurs very rapidly, because the roots of the phrenic nerve are involved, the intercostals being necessarily paralysed with the parts below. In some cases there is little immediate displacement, and the symptoms are slight until further displacement occurs in some movement. Thus a man who had met with an injury of this kind went to be shaved; during the proceeding, his head was turned on one side by the barber, with the unexpected result of causing displacement of the fracture, and immediate death. When the fracture is at the *cervico-dorsal* region, opposite the lower part of the cervical enlargement, the arms frequently escape at first, the early paralysis being confined to the legs and muscles of the trunk. Respiration is diaphragmatic only. After a few days the arms become involved, but their paralysis is often partial, affecting, for instance, only certain muscles, as the extensors of the hand, and it is often accompanied by local spasmodic movements. Movements and pressure cause pain, and there is local muscular rigidity. The head may be in normal or in abnormal position. Vaso-motor disturbance in the face and general hyperpyrexia have been observed. There is not unfrequently alteration in the joints, especially the knee-joints, apparently the result of the presence of fluid in them.

In fracture of the *dorsal vertebræ* (2—11) the arms escape, the legs are paralysed, and the trunk-muscles up to the height of the lesion. The pain in the trunk may be very severe. There is hyperæsthesia or anæsthesia in the parts below. The reflex action in the legs is excessive; that in the trunk is abolished at the level of the lesion. The *last dorsal* and *first lumbar* vertebræ are fractured more frequently than the others, and there result paralysis of the legs, complete or irregular, severe pains, tingling, &c., sometimes followed by hyperæsthesia or loss of sensibility, and by rapid disturbance of nutrition, in both the muscles and the skin. In fracture of the *lower lumbar vertebræ* the symptoms are often slight; below the extremity of the cord the nerves occupy a smaller space in the canal, and so may escape compression by a moderate displacement. If they suffer, the fractured vertebræ unite very slowly. A false joint is occasionally formed. Secondary myelitis and its consequences are frequent causes of death at a variable period after the injury.

CARIES OF THE SPINE.

Caries of the bones of the spine is a frequent cause of paraplegia. It is often termed "Pott's disease," from the English surgeon, Percival Pott, who first described it (in 1779) as a cause of paralysis.

Causes.—Males are said to be rather more liable than females, but the difference in sexual incidence is not great. It is more common in childhood (after three), and next in early adult life, but it may occur at any age, and is perhaps more common in the second half of life than any other scrofulous lesion. I have known it commence at fifty, and it has been met with as late as seventy. It is distinctly a manifestation of the tubercular and scrofulous diathesis, and evidence of such inheritance is to be traced in most cases. Occasionally the sufferer himself presents such indications, *e. g.* lung disease, or caries of other bones. It occasionally develops simultaneously with other signs of acute general tuberculosis. Injuries seem frequently to excite the bone mischief in the spine, as they certainly do analogous bone disease elsewhere, in those who are predisposed; possibly, sometimes, in healthy persons. Falls, blows on the back, and severe strains are the most frequent traumatic antecedents. The last may act by straining the ligaments and setting up inflammation, which spreads to the bones directly or through the intervertebral cartilages. There is usually an interval, sometimes of many months, between the injury and the definite symptoms of bone disease.

Other causes besides scrofulous disease have occasionally been known to cause breaking down of the bodies of the vertebræ with angular curvature and all its consequences. Soft growths in the spine and, in rare cases, syphilitic formations have had the same effect. Osteitis, due to extension from adjacent inflammation, is a rare cause; thus caries of the cervical spine has been secondary to a primary retro-pharyngeal abscess. The disease sometimes follows septicæmia, probably in consequence of a secondary septicæmic abscess in the bone. This was the case in a surgeon about sixty years of age, in whom symptoms of blood-poisoning followed an injury to the ankle. They were succeeded by paraplegia of rapid onset, and this by the development of angular curvature in the lower dorsal region. In a large number of cases no exciting cause can be traced.

Pathological Anatomy.—The changes in the bones need not detain us, since they belong to surgical pathology, except in so far as they lead to damage to the cord. There is inflammation of the bodies of the vertebræ, and of the intervertebral substance, often at first with enlargement, afterwards with breaking down of the substance of the bone, which gives way under the pressure to which it is exposed, so that deformity of the spine results. Products of inflammation and destruction of tissue, more or less purulent in character, accumulate either inside the canal or outside the vertebral column. In the latter situation they are increased in quantity by the inflammation they excite, and the well-known forms of abscess result.

Within the canal the inflammatory products have a firmer consistence. Although the inflammation extends to the loose cellular and adipose tissue between the bone and the dura mater, and often to the dura mater itself, it has little tendency to spread as a purulent meningitis, or even to penetrate the dura mater, the outer layer of which becomes irregularly thickened in the neighbourhood of the disease (see Fig. 93), while the inner surface of the membrane remains normal. The firm consistence of the inflammatory products, often caseous in character, is an important pathological feature, because this material is so often the agent by which the cord is compressed.

The displacement that results from the disease varies according to the extent and character of the disease. Occasionally there is simply a lateral displacement—one spine is a little to one side of that above it. More commonly the collapse of the bodies leads to "angular curvature," the spinal column is bent forwards at an acute angle, and one or two vertebral spines are much more prominent than the others. There is often, however, a less abrupt bend; the curvature may extend over four or five vertebræ. Less commonly one spine projects more than the others without any curvature.

The *nerves*, as they pass through the membranes and intervertebral foramina, are irritated by the inflammation, and often compressed by the thickening of the dura mater which sheaths them, and those passing by the seat of compression may also suffer from the narrowing of the canal. They may be found red and swollen, or shrunken and grey. They may be damaged when the cord is normal, or but little affected when this is compressed.

The damage to the *spinal cord* depends on the secondary consequences of the caries, and is variable and uncertain in both occurrence and character. The mechanism of the damage is twofold—compression and inflammation. The relation between the two is considered more fully in a subsequent chapter (Compression of the Spinal Cord). Slow compression may be attended by chronic inflammation, secondary not only in character but in course, or by an acute inflammation out of proportion to the compression, alike in degree of severity and in rapidity of development. It is important to recognise these differences. The mechanism of compression varies. The most frequent is the collection of inflammatory products outside the dura mater and the thickening of this membrane. Less commonly the cord is compressed by the displacement of the bone, or by fragments of bone that are pushed into the canal. Often both these causes are influential, as in the case shown in Fig. 92, in which the compression (at S) is between displaced bone in front and inflammatory products (E) behind. Probably an inflammatory swelling or abscess of the bone is sometimes the mechanism of compression, since the signs of pressure have disappeared when an abscess has formed outside the spine, or even when deformity has come on—the breaking down of the bodies having relieved the compression produced by their enlargement.

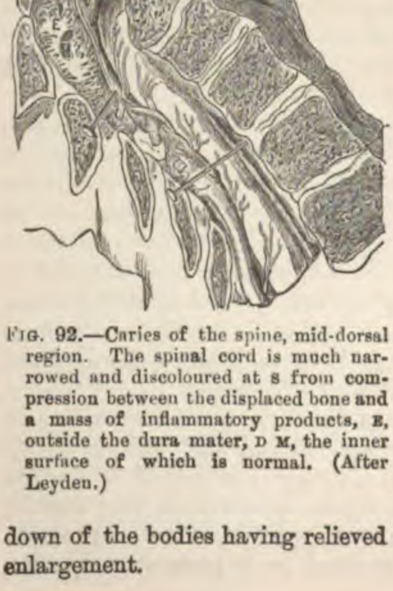

FIG. 92.—Caries of the spine, mid-dorsal region. The spinal cord is much narrowed and discoloured at S from compression between the displaced bone and a mass of inflammatory products, E, outside the dura mater, D M, the inner surface of which is normal. (After Leyden.)

SYMPTOMS.—Caries of the spine causes symptoms of three classes: (1) those of the bone disease; (2) the effects of damage to the nerve-roots; (3) those due to the changes in the cord itself. Only the symptoms due to the damage to nerves and cord come within the special province of this book, but the bone symptoms are of much importance, as on them the diagnosis of the cause of the nervous symptoms often depends. It is this which gives the subject its high medical importance, and brings the symptoms and recognition of bone disease into the province of practical medicine.

Symptoms of Bone Disease.—The first is pain in the spine, chiefly felt at the affected spot, increased by movement, and especially by pressure on the bone. The local tenderness is a very important sign. It is elicited both by direct pressure on the spines and by lateral pressure; if they are grasped and pressed to one side considerable pain is usually produced. Nevertheless tenderness is occasionally absent, not only in cases of slight character and doubtful nature, but also in those in which paraplegia has come on some time after local curvature. The increase of pain by movement is greatest when the disease is in the more mobile parts of the vertebral column, especially when it is in the cervical region. Movement of the head occasions pain, and there is an instinctive fixation of the head, which is sometimes inclined to right or left, less commonly backwards. It may thus produce the aspect of torticollis, which differs from that due to muscular contraction in that the sterno-mastoid is tense on the side towards which the head is turned, the muscle being simply stretched by the deviation of the head.

The deformity of the spine is a later symptom than the tenderness, and usually comes on gradually. Its characters have been already described. It is often absent when the disease is in the cervical region. In this part there is usually another symptom—thickening of the tissues about the spine, which is rare in other parts. In any region an abscess may form in the neighbourhood of the disease. Those which come backwards, or descend to the groin by the psoas muscle, can be recognised externally. Those that form in front of the diseased vertebræ may give rise to symptoms that are puzzling, if the existence of spinal caries is not known. Thus a retro-pharyngeal abscess may cause difficulty of deglutition, and one in the dorsal region may cause symptoms of œsophageal obstruction.

Increased pain and tenderness in the vertebral column often precede the indications of damage to its contents. The symptoms due to interference with the *nerve-roots* are very variable, and may be severe or absent; usually they are moderate in degree, and consist of pain on movement along the course and in the distribution of the nerves that emerge at the affected region, often hyperæsthesia of corresponding extent, sometimes with spots of anæsthesia. There is also muscular weakness and sometimes muscular wasting. These symptoms are more fully described in the chapter on Compression. Here it may suffice to [illegible] that they are conspicuous only in the minority of the cases, and chiefly occur when there is pachymeningitis. The motor symptoms are most marked when the disease is in the cervical region, and the nerve-roots for the muscles of the arms are damaged, since slight and partial impairment is more readily recognised in the arms than in the legs. If the disease is in the highest part of this region, the pains may be referred to the occiput. Herpes zoster has been occasionally [illegible] along the course of the irritated nerves. Spasmodic contraction in the muscles supplied by the affected roots is extremely rare in [illegible] Reflex action is abolished in the affected parts, and the change in the superficial reflexes of the trunk sometimes gives important diagnostic information. Disease of the lower cervical roots [illegible] symptoms of deranged action of the sympathetic on that side of the head, occasionally shown in the pupil, more often in the [illegible] I have seen persistent sweating on one half of the forehead [illegible]

[illegible] of interference with the functions of the spinal cord [illegible] due to impairment of its conducting power, causing [illegible] the lesion. As the disease is most frequently in some [illegible] region, paralysis of the legs is the common effect. [illegible] vary in their characters, according not only to the [illegible] disease, but also to the immediate process of damage [illegible] The signs of caries may have existed for years before [illegible] Angular curvature may even come on in early [illegible] and paralysis not till adult life More commonly the [illegible] the two is not long, varying from a few months to [illegible] but either may first be conspicuous. The cases are [illegible] in which caries is not suspected until the paralysis leads to [illegible] of the spine, and it is not uncommon for the signs of [illegible] to remain equivocal for some time after paraplegia has [illegible]

[illegible] symptoms vary much in their mode of onset. Usually [illegible] cause, but sometimes a strain of the back or [illegible] seems to excite a change in the condition of the [illegible] of which the cord suffers. It is easy to conceive [illegible] are favorable, a very slight strain may be [illegible] as the sneezing in the case mentioned below. [illegible] symptoms have commenced, they may develop quickly [illegible] of chronic onset may be mentioned cases in [illegible] reached a considerable degree of intensity at the [illegible] four months, and two months, after their com[illegible] onset is sometimes much more rapid; in one [illegible] paraplegia at the end of three weeks. Some[illegible] acute, and in such cases, when there is no corre[illegible] bone, acute myelitis set up by slight pressure [illegible] by which the cord suffers. Thus, in one [illegible] slight weakness existed for three weeks, and then the power of standing was lost in a single night. In another child, aged eight, who had presented for two years indications of disease of the cervical vertebræ, the power of moving the legs was lost in the course of twenty-four hours; during the second day the left arm became paralysed, and, at the end of a week, the right arm. Very rarely the onset is instantaneous, probably from sudden displacement. A child with angular curvature was walking across the room, when she fell, and on being lifted up the legs were powerless. The more rapidly compression occurs, the slighter is the amount necessary to abolish conduction.

Both legs usually suffer together; rarely one is paralysed before the other; very rarely one leg suffers much and the other little or not at all. Thus in one case angular curvature developed in childhood; at sixteen there was an attack of weakness in the legs, which passed away at the end of three weeks. At 17½ the patient sprained his back; pain in it followed, and six weeks later the right leg gradually became weak, and a year afterwards presented intense spastic paralysis, the left leg being very little affected. He ultimately recovered. An instance in which one leg became affected before the other is that of a woman, forty-five years of age, who suffered from pain in the spine, and one day, when walking, sneezed violently three times, and immediately felt "pins and needles" about the right knee, and presently in the foot. The leg became almost powerless during the next three days. A fortnight afterwards she felt similar tingling in the left knee, and at the end of another week in the foot, and during the next three weeks this also lost power, so that at the end of six weeks from the onset both legs were motionless. The diagnosis was verified on her death, six months later.

The characters of the paralysis in relation to the seat of the disease are described in the chapter on Compression. In the most frequent cases the dorsal cord is damaged, and whenever the compression is above the lumbar enlargement the condition of the legs is that of "spastic paraplegia." If it is situated in the cervical region there may be muscular atrophy in the arms, sometimes palsy without atrophy, according to the seat of the disease. When, as is commonly the case, the arms suffer from damage to the nerve-roots, they suffer before the legs, but if the disease is so high up that the arms suffer from the damage to the cord, the legs may be paralysed before the arms, as in the case of the child mentioned above. In this case the muscles of the shoulder were wasted, the disease being at the level of their nerves, while the forearm muscles were not wasted. In this case, as in others of similar seat, the diaphragm was paralysed. The muscular part of the spinal accessory may be involved, and the power of supporting the head may be almost lost. Very rarely, from the disease of the highest cervical vertebræ, other nerves of the medulla are implicated, especially the hypoglossal and the fibres of the spinal accessory for the palate.

Sensory symptoms due to the disease of the cord are less common than motor palsy. The onset of paraplegia may be preceded by subjective sensations in the legs. Dull aching pain in them is also not uncommon. Often there is no loss of sensation; in other cases there are various degrees of loss. Touch or pain may be impaired alone, or there may be absolute loss of sensibility up to the level of the lesion. Partial loss is more common. Reflex action in all forms is excessive (unless the disease involves the lumbar enlargement), and great excess of the superficial reflex action is a common and important feature. The legs are often cold, and sometimes perspire continually. The sphincters are often affected, sometimes early, but they may escape even where there is complete motor palsy of the legs.

Complications.—Among common complications are bedsores, cystitis, various secondary effects of the bone disease, such as abscess, local scrofulous disease elsewhere, and general tuberculosis. Tubercular tumours in the brain occasionally coincide with the caries. In very severe cases peculiar secondary mischief has occurred in the spinal cord, and has run an independent course, giving rise to very anomalous symptoms.* Thus a descending myelitis may invade the lumbar enlargement in its entirety and abolish its central and reflex functions, causing rapid wasting of the muscles and acute trophic changes in the skin. Inflammation may *ascend* the pyramidal tracts and thus paralyse the arms—an instance of the strange limitation of inflammation to functional tracts when it passes in a direction opposite to secondary degeneration. Ascending degeneration of the posterior median columns may spread to the postero-external columns, and cause symptoms of ataxy by invading root-fibres at a higher level. Ataxy may come on as the power returns, when the disease is in the dorsal region, probably by damage to the path from the muscles to the cerebellum. Lastly, myelitis may occur in disseminated foci in various parts of the cord, and even in the medulla, giving rise to scattered symptoms of anomalous character.

Course.—The bone disease may heal, union occurring between the altered tissues, or it may persist with continued formation of pus, or may become quiescent with occasional periods of renewed activity. The cord mischief is influenced by the state of the bone disease, although its progress may be to some extent independent. Thus inflammation in the cord, in excess of the compression, may subside in spite of the continuance of the bone mischief. Pressure on the cord may be relieved, although the bone disease continues, and even sometimes as a result of the increased breaking down of bone and exit of pus by another channel. A case is mentioned below in which paraplegia passed away as angular curvature developed. Hence there is no strict correspondence between the course of the bone mischief and that of the cord disease. In some cases the paralysis, motor and sensory, persists. More often the sensory loss passes away, while motor paralysis remains, usually as spastic paraplegia, and, in severe cases, flexor spasm comes on with muscular contractions. Life may be prolonged in that condition for years, but often, in such cases, bedsores form, or cystitis leads to kidney disease, or other tubercular disease develops and leads to death, or the lessened respiratory power renders an attack of bronchitis fatal. In many cases, again, the paralysis gradually passes away even when the compression continues, and the cord is found considerably narrowed if the patient dies from some other cause. It is possible that such compression is sometimes produced slowly without impairment of conduction. In children recovery occurs far more readily than in adults. Even in adults, however, recovery may occur from palsy that has lasted for more than a year, with all the signs of descending degeneration in the cord, amounting to severe spastic paraplegia. Usually rest on the back or mechanical supports are necessary to secure recovery, but it occasionally occurs without these measures. A youth acquired angular curvature at sixteen; at twenty paraplegia came on slowly, and progressed with some variations during the next seven years. There was then absolute motor palsy in the legs, and sensation was lessened. He refused to rest, and continued to follow his occupation, which was however a restful one, that of a tailor; he took cod-liver oil and iron, and gradually regained useful power, so as to be able to walk about. Such a case, however, is exceptional.

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* See Charcot, 'Leçons sur les Mal. du Syst. Nerv.,' tom. ii.

Relapses sometimes occur in cases that improve, although they are certainly not nearly so frequent as might be expected from the nature of the disease. In the majority of cases recovery, once obtained, is permanent. In a minority the paralysis returns when some exciting cause renews activity in the bone disease. The tendency to relapse and the possibility of repeated recovery are very strikingly shown by the following case. In a girl of fifteen, paraplegia developed during nine months, slowly at first, more rapidly towards the end of that time. She came under my care six months later, having been unable even to move her legs for that time. Bone disease had not been previously suspected, but there was slight tenderness and enough lateral irregularity of the lower dorsal spines to show the nature of the case. Rest in bed and tonics were soon followed by improvement; in six weeks she could stand, and in four months was able to walk well. As she gained power, angular curvature came on, prominence of the seventh and eighth dorsal spines. Five months after her discharge she fell and struck her back; the curvature increased, and her legs gradually became weak again. Seven months after the fall she was readmitted, unable to stand, although the paralysis was not absolute. There was foot-clonus on each side. Sensibility was lessened below the ensiform cartilage. Rest on the back was again followed by slow improvement. In three months she could just walk. She was then suspended, and encased in plaster of Paris. At the end of another month she could walk about the room, and no clonus could be obtained. She was soon

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[illegible] Mal. du Syst. Nerv.," tom. ii.

diagnosis occurs; the affection is apt to be mistaken for a primary disease of the cord or its membranes—a transverse myelitis when the dorsal region is affected, a progressive muscular atrophy, or primary pachymeningitis, when the disease is in the cervical region. A correct diagnosis can only be made in these cases by recognising the significance of the slight bone symptoms that are always present, the deep tenderness, and often slight irregularity. Even slight irregularity derives significance from tenderness, limited in extent and corresponding in position to the deviation. The irregularity may be absent at first, and then its development is doubly significant. An observed increase in the amount of displacement gives significance to even a slight irregularity.

The early excess of the cutaneous reflex action (from the sole, for instance), while not conclusive, adds weight to the other symptoms of bone disease. It may be very marked even while the patient is able to walk about. Distinct root pains are always of great significance, and this is increased by the detection of spots of anæsthesia along their course. All these symptoms derive additional weight from their coincidence in level with the spinal irregularity.

It is this which enables the pains to be distinguished from the condition for which they are most frequently mistaken—a trunk neuralgia. This error is especially common when the pain is chiefly unilateral. In all such cases the spine should be carefully examined, and any tenderness of the bone at the level of the pain should excite suspicion. I have also known unilateral abdominal pain due to caries to be mistaken for that of renal calculus.

When damage to the cervical roots causes muscular wasting in the arms, the case may be mistaken for one of progressive muscular atrophy, but differs in the distribution of the wasting, in the pains, and the impairment of sensation. These occur in primary cervical pachymeningitis, which is distinguished by the absence of the signs of bone disease, and in the wider extent through which the root symptoms extend.

In the first half of life the recognition of bone disease is practically tantamount to the recognition of caries. In the second half, however, the relative infrequency of caries, the greater frequency of growths in the bone, and the occurrence of eroding aneurisms, introduce a fresh diagnostic problem. The absence of any other indication of a tumour or an aneurism is the first distinction; and the second is the fact that in both these diseases the root pains commonly reach a degree of severity scarcely ever attained in caries, and are especially increased by movement.

When there are merely tenderness of the spine and slight weakness of the legs, the question may arise whether there is organic disease, or merely the condition termed "spinal irritation," or mere functional pain and tenderness and weakness of the legs. In these cases the tenderness is usually found over a considerable area of the spinal afterwards discharged, and her progress continued, so that, at the end of nine months, she could walk five miles, and there was no trace of clonus, although there was still some excess of the knee-jerk. She soon afterwards married, and bore a child, which died two years and a half after her discharge. She caught cold at the funeral, and a fortnight later again began to lose power; in six weeks the legs were almost motionless, with marked foot-clonus; sensation to touch was lost up to the umbilicus, that of pain being preserved. Neither rest nor encasement caused any improvement. After some months sulphide of calcium was given, and in a few days power began to return; in a month she could take a few steps, and in four months she could walk about the ward without difficulty. She made another good recovery. Some years later, however, paralysis again came on, and this attack proved permanent.

Several cases have come under my notice in which the subjects of caries in early life, which healed without damaging the cord, have at some period in adult life presented the symptoms of primary lateral sclerosis—simple spastic paraplegia, without any root symptoms or evidence of renewed activity of the bone disease. If there is a connection between the two, it is probable that the cord has suffered compression so slowly that its functions have not been interfered with, but, nevertheless, the vitality of the pyramidal fibres has been rendered less enduring.

It is important to remember that the deviation is often lateral,* and the chief difficulty is due to the fact that a slight lateral deviation or slight prominence is not unusual in normal spines Hence it is important for the student to make himself familiar with the degrees of deviation that occur in health. At the same time it must not be forgotten that a deviation not greater than occurs in health may be due to disease. If it coincides with distinct tenderness, and especially also with the position of root symptoms, it may be accepted as evidence of disease, probable or certain, according to the character of the symptoms.

The pathology of the affection only concerns us so far as it relates to the effect on the spinal cord, and this is considered in the chapter on Compression.

Diagnosis.—When clear indications of caries precede the paralysis, the nature of the case can hardly be mistaken. The obvious inference, that the affection of the cord is secondary to that of the bone, is scarcely ever wrong. When the two develop together, mistakes in diagnosis are often made, but are usually due to the want of *repeated* examinations of the spinal column. It is when the root or cord symptoms precede distinct evidence of bone disease, and when the latter is so slight as to be equivocal, that the chief real difficulty in diagnosis occurs; the affection is apt to be mistaken for a primary disease of the cord or its membranes—a transverse myelitis when the dorsal region is affected, a progressive muscular atrophy, or primary pachymeningitis, when the disease is in the cervical region. A correct diagnosis can only be made in these cases by recognising the significance of the slight bone symptoms that are always present, the deep tenderness, and often slight irregularity. Even slight irregularity derives significance from tenderness, limited in extent and corresponding in position to the deviation. The irregularity may be absent at first, and then its development is doubly significant. An observed increase in the amount of displacement gives significance to even a slight irregularity.

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* To discover lateral deviation it is well to make an ink-dot on the skin over the middle of the tip of each spine, care being taken that the skin is not stretched to e side. The ink-dots may be copied on tracing-paper.

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When there are merely tenderness of the spine and slight weakness of the legs, the question may arise whether there is organic disease, or merely the condition termed "spinal irritation," or mere functional pain and tenderness and weakness of the legs. In these cases the tenderness is usually found over a considerable area of the spinal column, with more than one point of special intensity; it is superficial as well as deep, and may change its seat; there are no root pains or spots of anæsthesia. There is more danger that caries of the spine in a young woman may be passed as hysterical paraplegia than of the opposite error. Especially when the subjects of caries present distinct symptoms of hysteria, there is risk, as experience shows, that unequivocal symptoms of caries may be overlooked. Some other diagnostic indications are mentioned in the chapter on Compression.

PROGNOSIS.—Our ignorance of the precise character of the morbid process which is damaging the cord renders the prognosis, in every case of caries, a matter of much uncertainty. Nevertheless there is no disease of the cord in which symptoms of equal gravity so often pass away. The cases are few, therefore, in which hope is unjustified, but they are equally few in which we are warranted in a confident expectation of recovery. In childhood the prospects of recovery are certainly better than in adult life, and they are least in declining years. Damage to the cord between the enlargements is less serious than when these are affected, because the strong tendency to trophic changes constitutes a grave danger when the lumbar enlargement is diseased, and the diminished breathing power an equally serious danger in disease of the cervical enlargement, especially when this is high enough to entail the additional danger of paralysis of the diaphragm.

But how perilous a condition may be recovered from is shown by the fact that the child mentioned on p. 291 had paralysis of all four limbs, the diaphragm, and weakening of the intercostals, and yet recovered. Still more striking is a case narrated by Dr. Buzzard, in which disease in the region of the third cervical vertebra caused almost complete palsy of arms, legs, intercostals, and diaphragm, respiration being carried on by the accessory muscles of the neck. Yet the child recovered in spite of the occurrence of an attack of pneumonia when the paralysis was at its height. A girl of thirteen, whose cervical caries was accompanied by all the symptoms of cerebellar tubercle, recovered. All these, it will be noted, were children. Neither rapidity nor slowness of onset affords any guide to prognosis, nor does the relative order of paralysis and curvature, or the degree of palsy. Severe spastic paraplegia may pass away entirely, provided it remains extensor in character. The prognosis is perhaps a little better when there is no loss of sensation, since this proves that the damage to the cord is moderate in degree; but even complete anæsthesia does not preclude recovery, as the cases mentioned show. The danger to life is dependent in considerable degree on the evidence that the scrofulous or tubercular tendency is active elsewhere, and also on any difficulty in securing proper treatment. The prognosis is, moreover, still in a transitional state on account of the uncertainty regarding the range of successful surgical treatment.

to rest, and may be effective even alone, as is shown by the case mentioned on p. 293. But rest should always be secured if possible.

Counter-irritation, opposite the seat of caries, has sometimes appeared to do good, perhaps acting especially on the process of myelitis which attends compression. The actual cautery, in mild form, is that which has been most frequently found useful. Instances of speedy improvement are on record, but it is not always effective, nor can indications for its use be laid down. Sulphide of calcium, which is said to influence scrofulous processes, deserves further trial, although I have not found it so apparently effective in any other case than that mentioned on p. 294. Benefit can only be expected from its use when, as is often the case, inflammatory products constitute the compressing agent. Extreme care in general management, to avoid bedsores, bladder trouble, and bronchitis, are of great importance. In many cases there is a tendency to improvement after a time, especially when the bone disease is stationary, or the cord has been inflamed, and in these the mere maintenance of life may result in recovery of strength. When power has returned, but the use of the limbs is restrained by the muscular contractions which come on during the paralysis, the ability to stand and walk may be quickly restored by tenotomy, and more slowly by the use of extending splints, &c. Electricity is useful only to maintain the nutrition of the muscles when these are wasting, in consequence of damage to their nerves.

The belief expressed in the first edition of this work, that the cases are numerous in which relief to the compression may be afforded by the surgeon, has been fully realised. The path was, indeed, being then opened up by McEwen of Glasgow, who has shown, with others, especially Victor Horsley, how safe and successful the operative treatment is in properly selected cases; and these, indeed, include a large proportion of the cases in which other measures have failed to give relief and must probably have remained unsuccessful. The operation is contra-indicated in cases of rapidly advancing general tuberculosis (to which most deaths after operation have been due, and also, as a rule, in children, in whom other measures are, as has already been stated, usually successful) When the indirect consequences of the disease have greatly reduced the patient's strength, the question of an operation may still be entertained if it is a distinct alternative to a speedy death, provided the peril is such as a restoration of the functions of the cord might possibly remove. On the other hand, the operation ought not to be regarded as the first resort in the majority of cases. The large number of cases that recover under the treatment described above make it certain that, in most cases, these measures should first be tried; and this conclusion is the clearer since the duration of paraplegia, for a year or more, seems to interpose no appreciable barrier either to recovery or to the relief that can be given to the pressure. On the contrary, indeed, the prospect of recovery is better if the bone disease has become quiescent, or the caries has actually healed, processes that must be furthered by the preliminary rest. The operation of laminectomy has the best prospect of success in the cases, fortunately very numerous, in which the products of inflammation outside the dura mater compress the cord. These, whether caseous material or a mass of connective tissue into which the inflammatory products have been changed, can be readily removed without opening the dura mater. Even if displaced bone is the source of pressure, the removal of the arches affords relief to it. The chief obstacle to success is the extent to which myelitis has proceeded out of proportion to the compression. This, necessarily, no removal of the pressure can relieve. In proportion as the inflammation has been acute without evidence of rapid compression, relief is unlikely. But this is not the only cause of a rapid onset, and a difficult question arises in such cases, whether the mechanism is not such that an immediate operation is justified. It may be due to sudden displacement of bone or to sudden escape of pus into the canal, as well as to myelitis, and in the two former cases it is possible that relief may sometimes be given, and an immediate operation may be wiser than delay. An operation is suggested whenever a sudden increase in the curvature or severe root-pains coincide with the acute onset of paralysis. Without these symptoms, the rapid onset is probably due to acute myelitis, and then an operation cannot be expected to lessen the interference with the conducting fibres. It is also called for in cases in which the disease is high up, and by its interference with the respiratory mechanism is threatening life. To justify surgical interference, however, the existence of caries must be beyond doubt. The operation involves a small danger to life, although it may not be great, and this fact must always receive due consideration in regard to the certainty of diagnosis as well as the prospect of relief.

TUMOURS AND OTHER DISEASES OF THE SPINE.

Growths in the Spinal Column.

The bones of the spine are sometimes the seat of primary or secondary growths, and less commonly are invaded by tumours springing from the various structures and even organs in front of the spine. They may also undergo changes as the result of the obscure condition known as osteomalacia, and these changes may occasion pressure on the cord, and so give rise to paraplegia.* Cancer (scirrhous

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* Köppen, 'Arch. f. Psych.,' xxii.

[illegible] and sarcoma are the most common forms of growth; [illegible] has been also met with. Cancer is sometimes secondary to a primary growth elsewhere, in the breast, stomach, &c. A primary [illegible] usually begins in the bodies of the vertebræ, and spreads from [illegible]. The bodies may collapse, because the soft tissue [illegible] under the weight it has to bear, and [illegible] may occur, or some other abrupt deviation [illegible]. From the bodies the growth may extend into [illegible], and narrowing the intervertebral [illegible] which the nerves pass. It may extend into the [illegible] the spines, and may also grow into the muscles [illegible] round the vertebral column. The nerve-roots [illegible] simple inflammation, (3) sometimes by [illegible]. They may thus be found reddened, swollen and [illegible] enlarged and hardened. The growth [illegible] spread in the adipose tissue between the [illegible] and may even entirely surround the dura [illegible] suffer compression, but this is less [illegible] when curvature occurs. On the other [illegible] acutely: considerable inflamma[illegible] far more frequent than in caries. The [illegible] of the dura mater by the [illegible] acute local inflammation is [illegible] spread in the cord upwards and [illegible] invades the cord itself.

[illegible] are the same as those of similar [illegible], more frequently than [illegible] together, are most common [illegible]. An injury has been supposed [illegible] other situations.

[illegible] of the presence of the [illegible] occasionally absent; local [illegible] with movement of the vertebral [illegible] is never an early sym[illegible] swelling may be felt on one [illegible] when the disease is in the [illegible] perceptible in the posterior [illegible] which sometimes occurs [illegible] its external characters, except [illegible] than a sharp angle, and [illegible] of a tumour, never by [illegible]

The [illegible] almost invariable in spinal growths, and [illegible] are the most distressing, and also the [illegible] radiating pain in the course of the nerves [illegible] due to their irritation by pressure or [illegible] are far more prominent symptom in this disease than in caries. At first slight, they gradually increase to extreme intensity. It was this feature that led Cruveilhier to call the disease *paraplegia dolorosa*, a name it has since commonly borne. The pain at first intermits, but subsequently is constant, with paroxysms of greater suffering from time to time. It is usually a sharp and lancinating pain, and its special characteristic is the degree in which it is increased by movement. Even slight movements of the trunk induce the paroxysms. The distribution in the arms, trunk, legs, depends on the seat of the disease. It is extremely rare for these pains to be absent, but they occasionally occur late instead of early.[*] On the other hand, they may precede other symptoms for months. Cutaneous hyperæsthesia usually accompanies them, and spots of anæsthesia often develop, after a time, in the areas to which the fixed pains are referred. Corresponding damage to the motor roots may cause painful muscular contracture (Figs. 56 and 57, p. 112), paralysis, and wasting. Contractures may also occur in the parts below the disease, produced by the persistent irritating pressure on the motor tract in the cord. Paroxysms of spasm often attend the pains, especially in the abdominal muscles when the disease is in the dorsal region, and are apparently reflex in nature.

The damage to the cord causes symptoms similar to those in caries, and described more fully in the chapter on Compression. The chief difference from caries is the greater frequency of a rapid onset of the paraplegic symptoms, due to the invasion of the cord by acute inflammation. All power in the legs is often lost in twelve or twenty-four hours, and this when no curvature has taken place. Such acute paraplegia is not uncommonly preceded for a week or so by retention of urine, probably indicative of slight pressure on the cord. Displacement of bone has also been known to cause rapid paralysis.[†] On the other hand, especially in very slowly growing tumours, the onset of the palsy may be gradual. I have known it to occupy several years in reaching a considerable degree. Probably in these cases the mechanism is a simple slow compression. Thus, compared with caries, the onset is more often rapid, and occasionally much more deliberate. The characters of the resulting paralysis are, as a rule, similar to those in caries, but are more frequently modified by the spread of inflammation, immediate or subsequent. Hence the central and reflex functions of the lumbar enlargement, although the bone disease is some distance above it, may be lost at the onset, or they may be at first normal or excessive, and afterwards lost. In a man with a growth in the mid-dorsal region, paraplegia came on rapidly, evidently from myelitis, and was followed by foot-clonus, &c. But a few weeks later the clonus suddenly ceased, the muscles became toneless, with loss of faradic irritability, and the skin began to slough; the inflammation had spread down into the lumbar enlargement. In many cases, however, there is no descending inflammation, and the reflex actions are in persistent excess, so that spastic paraplegia results. Sensation is lost rather more frequently than in caries. Other symptoms are the same as in compression from any cause. The course of the disease is from its nature progressive; occasionally, however, some improvement succeeds a rapid development of palsy, and is due to the partial recovery of a cord damaged disproportionately by inflammation. It is seldom, however, that life is prolonged for a sufficient length of time for this to occur. Much more rarely the pains lessen, although the growth spreads—perhaps from destruction of irritated nerves.

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[*] The pains succeeded curvature in a case described by L. Humphry ('Lancet,' 1884, i, p. 15).

[†] Humphry, loc. cit.

The *duration* of the disease varies according to the nature of the growth. In cancer it is to be measured by months. In slowly growing tumours the symptoms may last for years. Death may be due to bedsores, &c., to cystitis and kidney disease, to growths elsewhere, or the patient may be simply worn out by the prolonged agony. In one curious case a growth from an intervertebral cartilage caused characteristic symptoms for thirteen years, and then death by meningeal hæmorrhage.*

DIAGNOSIS.—The recognition of the disease is only a matter of certainty when signs of a tumour are present, but the probability almost amounts to certainty when such symptoms as those mentioned follow a primary growth elsewhere. Such pain as that above described, occurring in a person from whom a malignant tumour has been removed, should always be regarded with grave suspicion, and the grounds for this are not lessened by either an interval of several years since the removal of the tumour, or by the completeness with which this was effected. Especially is this true of cases of mammary cancer. The pains should lead to careful and repeated examination of the spine and a search for any abrupt deviation, of any kind, and in any part of the vertebral column; while the cervical spine should be examined at the sides and from the posterior triangle of the neck, the character of the bony prominences on the two sides being minutely compared. Indications of a growth may often be thus discovered long before they are obtrusive. It is also important to remember the fact that acute transverse myelitis may be an early effect of the disease, and if this lesion, in such a subject, coincides with marked local tenderness, the probability of secondary growth in the bone is very great; it is rendered still greater by preceding pains, and certain by coincident deformity.

It must be remembered that similar root symptoms are sometimes due to an aortic aneurism, or to a growth in front of the vertebral column, commencing, for instance, in the glands, and irritating the nerves as they emerge from the intervertebral foramina.

From intercostal neuralgia, the influence of movement on the pain is usually a sufficient distinction, even when cord symptoms are absent. The commonly bilateral character of the pain is a further difference. The symptoms of a growth in the lower cervical region are sometimes very closely simulated by symptoms which, from their character and course, are probably due to radicular neuritis. There are severe root-pains, referred to one arm (or less commonly to both arms), increased by movement and without marked tenderness of the plexus of nerve-trunks. The subjects are also in the later period of life. The distinction rests on the absence of the signs of a growth, on the slighter effect of motion on the pains, on the absence of any progressive tendency, and on the common presence of a history of gout, and especially on the absence of any symptoms of compression of the cord even after the nerve-roots have suffered for some months. Sometimes it is necessary to wait and watch the course of the symptoms. It is necessary to be very careful in assessing the value of slight irregularities; the vertebra prominens is apt to be thought to project too much even for its designation, and a deep bilateral swelling is readily assumed, under normal conditions, when the head is bent forward.

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* Boivert, 'Arch. de Phys.,' 1887, No. 8.

The chief difficulty in diagnosis is the distinction from caries when distinct evidence of a growth is absent. In the first half of life caries would alone be thought of in such a case, but in the second half the two diseases are about equally frequent. One distinction—suggestive, not absolute—is the intensity of the pain in tumour, taken in conjunction with its great increase when the patient moves. It is true that the root-pains of caries are said to be sometimes most severe, but such severity is not frequent enough to destroy the significance of intensity. I have not, for instance, seen a single case of caries (of a large number) in which the pain was comparable to that in most of the cases of growth that have come under my observation. Therefore, while absence of pain is of slighter diagnostic value (in favour of caries), great severity, and agonising increase by movement, are strongly in favour of vertebral growth. In each disease there may be angular curvature; but this, in growths, is usually soon succeeded by other signs of tumour. In caries these signs are absent, and an abscess often develops. A history or indication of tubercle is almost conclusive evidence of caries. These points will, I believe, avail for the distinction in most cases. In a few it is necessary to wait and watch before an opinion can be formed.

Among other diseases with which these growths may be confounded is the dorsal form of tabes, in which severe radiating pains occur in the trunk and not in the legs. But the wide extent of the root symptoms, the slight effect of movement, and the fact that the knee-jerk is lost, suffice for the distinction. There is also usually more extensive disturbance of sensation on the trunk. The distinction from tumours of the spinal cord and membranes is considered in a later chapter.

PROGNOSIS.—The prognosis scarcely requires formulating. The chief differences are in the time that life is likely to last. The pains usually persist, in spite of the progress of the disease, although there is a bare possibility of their subsidence. The chance of any return of power in the paralysed part is small, although not quite absent if the palsy develops in a manner to suggest a secondary myelitis, and the progress of the growth itself is slow.

TREATMENT.—Possibly in a few cases a growth may be so placed as to be removed, and an exploratory operation would be justifiable in any case that might prove suitable; otherwise treatment must necessarily be confined to the relief of pain, and to the avoidance of bed-sores and other results of the cord disease. Morphia is alone powerful for the relief of pain, but unhappily the dose has to be quickly increased, and the power of the drug is lessened by custom. It becomes a race between dose and pain, in which, if life lasts long, the pain not uncommonly gets in front of the narcotic. Cocaine, however, affords some relief in many cases, and may, with other anodynes, at least prolong the influence of moderate doses of morphia.

VERTEBRAL EXOSTOSES.

Exostoses sometimes grow from the bodies of the vertebræ into the spinal canal, and may compress the cord or nerves. They are, however, exceedingly rare. The symptoms may be those of slow compression of the cord, or of irritation, expressed chiefly by pain. They usually resemble those of a tumour of the cord or membranes rather than of the bones, but the pain is occasionally much increased by movement. Their chief characteristic is extreme chronicity. In one case the patient suffered frequent intense paroxysms of pain in the right groin, which had persisted for two years, with occasional intervals of freedom. There was some weakness of the legs, but no considerable paralysis. Ten years before, he had had some loss of sensibility in each thigh, which had passed away. An intra-spinal tumour was diagnosed; the post-mortem examination revealed exostoses from the bodies of the ninth and tenth dorsal vertebræ, slightly compressing the cord. Although extreme chronicity may raise a suspicion of exostosis, it is doubtful whether a confident diagnosis is ever justified except in the cases in which there are similar exostoses elsewhere. This rare indication existed in a patient, under the care of my colleague Dr. Barlow, who presented multiple exostoses, and paraplegia of gradual onset, which was supposed during life, and found after death, to be due to a similar exostosis within the spinal canal. It had sprung from one of the lumbar vertebræ, and had compressed the nerves of the cauda equina.

Exostoses constitute a more promising field for the surgeon than other kinds of vertebral tumour. Many of them are so placed that [illegible] removal is feasible. If situated in front of the cord, the [illegible] of some nerve-roots, at least in the dorsal region, might [illegible] access to the growth.

SYPHILITIC DISEASE.

Syphilitic caries of the bodies of the vertebræ is a rare variety, the symptoms of which do not differ from those of the scrofulous form. It has been observed in the cervical region, secondary to deep syphilitic ulceration of the pharynx. Nodes of the vertebræ, within the canal, are occasionally presumed to exist and to compress the cord, but I do not know of any pathological observation confirming the assumption, and it is probable that most of the supposed instances have been cases of syphilitic gummata in the meninges. Deep-seated thickening of the tissues about the cervical vertebræ sometimes occurs in syphilitic subjects. It may develop on one side or both, and is apparently due to a syphilitic cellulitis. The swelling may be felt either on each side and behind the upper cervical spine, or deep in the posterior triangle of the neck. It may damage the nerves before they enter the brachial plexus, causing a defined palsy, as in one case of the lower arm muscles. Movements of the neck may be interfered with, and irritation of the nerves may cause neuralgia-like pain, generally felt down the arm, and often very severe. Except by the absence of nodulation, it is scarcely to be distinguished from a deep-seated growth. The spinal cord does not usually suffer. All the symptoms pass away under antisyphilitic treatment. In one late case, however, iodide had no influence, although mercury quickly cured.

EROSION BY ANEURISM.

Bones, like other structures, may atrophy and waste before the pressure of an aneurism, and the bodies of the dorsal, or rarely of the lumbar vertebræ may be thus eroded by aneurisms of the aorta. The pressure and absorption take place from the left side. Two or three vertebræ usually suffer, and the bodies more than the intervertebral cartilages. The periosteum becomes thickened, and may resist the pressure and to some extent protect the cord. Sometimes, however, the cord becomes compressed, or the periosteum may come to form part of the wall of the aneurism, and may give way before the blood-pressure, so that rupture occurs into the spinal canal.

The symptoms vary much. Pain along the nerve-roots is usually severe, but this may attend aneurisms that merely compress the nerves after their emergence, and do not damage the bone. The process of erosion is usually attended by severe pain in the spine. But in the case of a patient under the care of Dr. Hughlings Jackson, an aneurism of the arch of the aorta had eroded the spinal column, caused pressure myelitis, which gave rise to complete paraplegia and paralysis of the sphincters, without causing any pain. The patient died of kidney disease secondary to cystitis. When the cord is reached, compression causes the usual paraplegic symptoms, of slow or rapid onset. Rupture into the canal is attended by sudden complete paraplegia and death, either immediate, or in the course of a few hours, from ascending paralysis, due to the hæmorrhage around the spinal cord.

The diagnosis is scarcely possible unless other indications of aneurism are detected, since the symptoms closely resemble those of a growth in the bone. The nature of the disease may, however, be suspected if such symptoms as have been described are succeeded by sudden paraplegia followed quickly by ascending paralysis.

HYDATID DISEASE.

Hydatid cysts sometimes develop in the loose adipose tissue between the dura mater and the bone, and it is believed that they sometimes form in the substance of the bones themselves. About a dozen cases have been collected by Leyden.* As the cyst grows, the bone of the arches sometimes becomes atrophied by pressure, and the cyst may develop outside the canal, so that there is a double cyst, outside and inside, connected by a narrower part. Occasionally the cyst develops also in front of the spine, in the thorax or abdomen. The internal cyst necessarily compresses the spinal cord, which often also becomes inflamed. The usual paraplegic symptoms develop, both motion and sensation being usually lost. Radiating pains along the nerve-roots are frequent. The symptoms, in themselves, resemble too closely those caused by other diseases of the spinal column to permit a diagnosis to be made, unless similar disease elsewhere suggests the nature of the spinal lesion, or unless the cyst can be felt in the back, when a puncture may prove its nature. All the cases hitherto recorded have terminated fatally, but if removal can be effected a cure may be possible. In one case, however, a girl aged twenty-two, in whom a small tumour beside the last dorsal and first lumbar spines was opened (on account of complete palsy of legs, bladder, and rectum), a continuous discharge of echinococcus cysts occurred, more than a hundred escaping in the course of four months, when the patient died in consequence of ascending damage to the cord. The tumour had been noticed for eight years before death, during four of which it produced symptoms of irritation of the nerves, and finally prominence of the vertebral spines developed.† But in a case under my care, with symptoms of an intra-spinal tumour in the lumbar region, Mr. Horsley trephined the spine and found a quantity of hydatid cysts compressing the cord. These were removed, and the patient recovered, but the damage was too great to allow the recovery of the extensive atrophic paralysis produced in the right thigh and hip muscles.

Numerous small cysticercal vesicles have also been occasionally found within the dura-matral sheath. In one case several such vesicles were attached to nerve-roots, one to each, in the lumbar region, while a considerable number on the cervical enlargement were beneath the pia arachnoid.* Such cysts may compress the cord, producing paraplegia, and even death. As in other forms of compression, the paraplegia may come on suddenly.†

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* 'Klinik der Rückenm. Krankh.,' Band i.

† Pedjkow, 'Med. Obos.' (Russ.), xxviii; and 'Cent. f. Nerv.,' xii, 271.

DISEASES OF THE ARTICULATIONS.

LATERAL CURVATURE OF THE SPINE seldom affects the functions of the cord. Even when slight compression has occurred, the slowness of its production has apparently prevented interference with function. In very rare cases some weakness of the legs has been present, possibly, but not certainly, the result of the curvature. Occasionally the intervertebral foramina have been narrowed, and the pressure on the nerves has caused radiating neuralgic pains.

VERTEBRAL ARTHRITIS.—In chronic rheumatoid arthritis the intervertebral articulations are sometimes involved. The symptoms are local pain, tenderness, and limitation of movement, which may go on to absolute fixation, especially in the cervical region, if anchylosis occurs. I have known the whole neck to be rigid from this cause, in a case in which also the movement of the lower jaw was much reduced. More frequently, movement is restricted by pain before the mechanical limit is reached. Sometimes an occasional clicking sound occurs. Enlargement of the ends of the bones may occur, and, in thin persons, may even be felt. The pain is increased by movement, and often by changes in the weather, and by fatigue. It is often felt through a considerable extent of the spine, and may extend to the back of the head. The cord is scarcely ever compressed, but the narrowing of the foramina may damage the nerve-roots. The articular processes, it will be remembered, form one side of the intervertebral foramina, which are thus encroached on by any enlargement of the bones. The nerves may thus be compressed and irritated, and radiating pains are produced, sometimes even a descending neuritis. The nerve-trunks in the limbs may then become tender as well as painful; muscular atrophy may even occur, and, when the disease is in the cervical region, there may be symptoms of disturbance of the sympathetic. Spasmodic torticollis is said to have been caused by this disease in the cervical region. The atlo-occipital articulation has been affected alone, or with the adjacent vertebræ, and peculiar interference with the movement of the head has resulted. Sometimes the foramen magnum is narrowed, or the odontoid process projects into the canal. Symptoms of damage to the nerves of the medulla have also been produced (Solbrig). In the dorsal region the symptoms produced are slighter in degree, but similar in general cha-

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* Hirt, 'Berl. kl. Wochenschr.,' 1887, No. 3. The relation of the symptoms to the parasitic disease is doubtful in a degree that makes it useless to mention them.

† As in a case recorded by Wiegand ('Warsaw Med. Ob.;' and 'Cent. f. Nerv.,' 1888, 665).

death, [illegible] preponderate over those
paralysis [illegible] spine may even become rigid.
The [illegible] process causes symptoms varies,
aneurism [illegible] and it has occasionally been
growth [illegible] without being suspected
suspected [illegible] distinct evidence of a rheu-
sudden [illegible] obvious arthritis elsewhere.
[illegible] indication as to the cause of
[illegible] remembered that the vertebral
[illegible] it may cause symptoms during
Hyd[illegible] is unsuspected. It occurs in both
between [illegible] in whom the symptoms are
sometimes [illegible] treatment is the same as that of
a dozen [illegible] alone needing special treatment,
the bone [illegible] on diseases of the nerves. The
the cyst [illegible] the malady is its diagnosis, because
cyst, [illegible] regarded as unconnected with local
the cyst [illegible] far more serious than it really is,
The inter[illegible] chronic meningitis or malignant
also [illegible]
motion [illegible]
nerve-[illegible]
closely [illegible]
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nature of [illegible]

[illegible]NES OF THE SPINAL CORD.

when a [illegible]
have term[illegible] membranes of the spinal cord has
possible. [illegible] The pia mater closely invests the
small tum[illegible] loose sheath around it, and the two
(on [illegible] and membranous expansions of
tinuous di[illegible] the "subarachnoid space." Each
escaping in [illegible] by a layer of epithelium. The
quence of [illegible] continuous with this membrane; the
for eight [illegible] sheath, is continuous with both the
ptoms of [illegible] vessels of the cord ramify, as we
vertebral [illegible] the lymphatic canals of the two are
ptoms of [illegible] spinal fluid within the vertebral
trephined [illegible] space, but there is a little fluid
the cord. [illegible] arachnoid. Both membranes are
damage wa[illegible] the pia mater are the more abun-
paralysis [illegible]

Numerou[illegible] the membranes are chiefly three
found with[illegible] inflammation. Tumours of the mem-
vesicles [illegible] in connection with those of the
region, whil[illegible] morrhage will be here described.

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* [illegible]

† [illegible]

an acute inflammation, which may spread widely. An ascending neuritis has been supposed in some cases to set up local inflammation. Suppuration adjacent to the vertebral column is sometimes associated with a general acute purulent meningitis, and has generally been regarded as the cause of the latter; but careful observations have rendered it probable that in most cases the external suppuration has been the consequence, and not the cause, of the meningitis. It is certain that acute general external meningitis occurs as a primary disease, and may run an intensely rapid course with profuse suppuration between the membrane and the bone. The subjects have generally been young, ill-nourished adults, and when any exciting cause has been traced, this has usually been exposure to cold. In some cases the pus has worked its way backwards between the vertebral arches (sometimes through the foramina), and has spread among the muscles of the back, sometimes forming also collections at one or more spots, either at the back or in front of the spine,—under the pleura, for instance, or behind the peritoneum. Such local collections of pus were also formerly regarded as the cause of the meningitis. It is still possible that this mechanism is effective in some instances, as when a retro-pharyngeal abscess is followed by the meningeal affection; but it is certain that in many cases the actual relation is the reverse, and that the external suppuration, in acute cases, is often secondary.* Hence it is probable that this is true of all cases in which the evidence of primary external disease is not conclusive. At the same time further observations are needed to decide the point.

Pathological Anatomy.—The inflammation is sometimes simple, but much more frequently purulent or semi-purulent. If simple, the dura mater may be merely reddened and opaque, with a little lymph on the surface; when purulent, the surface is covered by a layer of pus. In the more common semi-purulent form, such as is common in caries of the spine, a layer of inflammatory products covers the outer surface of the dura mater, soft, semi-caseous or firm caseous material, sometimes with liquid pus here and there in the firmer substance. Often the inflammatory products undergo development into fibroid tissue, a mass of which then lies between the dura mater and the bones. Such a layer, caseous or fibroid, may sometimes be half an inch thick (see Fig. 92, p. 288). It may either surround the dura mater, or be chiefly on one side. Sometimes there is an irregular nodular thickening of the outer layer of the membrane (Fig. 93). In most cases the inner surface is normal, even when the cord itself is inflamed. Occasionally the inner surface of the dura mater is also inflamed, and then the internal membranes are involved and adhesions form between them.

In all forms of external inflammation, the fat outside the dura mater quickly becomes absorbed, and the membrane may ultimately become adherent to the bone, or connected with it by intervening fibrous tissue of inflammatory origin.

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* Two instructive cases are recorded by Dr. W. H. Spencer, 'Lancet,' 1879, vol. i, and Dr. Maguire, ib., 1888, vol. ii.

The vertical extent of the disease varies much; when secondary to caries the inflammation is usually limited to the neighbourhood of the bone disease. When acute and general, as in the primary form, the space between the membrane and the bone is filled with pus through the whole length of the canal. Occasionally the pus only lies behind the membrane, between it and the arches of the vertebræ, apparently determined in position by gravitation. It ceases in the upper cervical region, where the dura mater is in closer contact with the bone, its ascent being perhaps also hindered by gravitation as the patient lies with the head raised. As already stated, pus is often found among the muscles of the back (having escaped between the arches), and sometimes collections are found in front of the spine. The amount outside is no doubt increased rapidly by the local suppuration excited by the presence of the pus. In these cases the membrane itself is swollen, and has a sodden aspect, resembling a piece of wash-leather, being softened in proportion to the duration of the affection. The inner surface is usually normal, and the pia mater is either unchanged or merely congested.

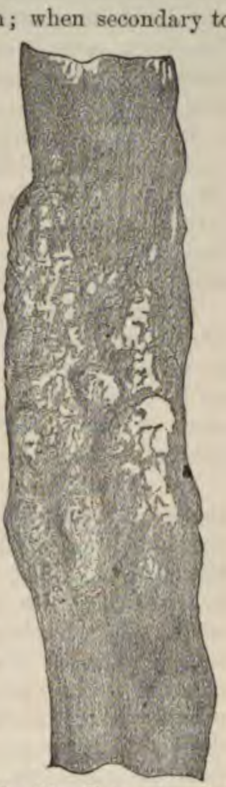

FIG. 93 —External pachymeningitis; nodular thickening of outer layer of dura mater in caries of the spine.

SYMPTOMS.—In different cases of external meningitis the symptoms vary much, and they are usually complicated with those of the cause of the inflammation. Those that occur in caries have been already described. In most acute cases the symptoms are nearly the same as those of internal meningitis, although they are subject to greater variations, and more frequently have unusual and anomalous manifestations, presently to be described. The chief symptoms are pain in the back, often referred to the loins, increased by movement, accompanied by stiffness of the muscles of the spine, severe root-pains, and cutaneous hyperæsthesia. The pains are usually associated with spasm; the hyperæsthesia may be followed by anæsthesia, and sometimes by paralysis of the muscles supplied by the irritated and damaged nerves. When there is a rapid formation of pus, the functions of the cord itself are rapidly interfered with in consequence of its compression, and the symptoms of irritation are to a large extent replaced by those of paralysis, beginning in the legs and ascending.

The motor palsy is accompanied by flaccidity of the muscles and loss of all reflex actions. Sensation may also be lost, or may be preserved or changed. The sphincters are usually paralysed. Bedsores form rapidly if life is sufficiently prolonged. A peculiar lividity of the skin has been sometimes noted. In these acute cases there is much constitutional disturbance, rigors, profuse sweating, and high fever, in one case amounting to 110° (Maguire).

In local secondary forms the symptoms differ in character according to the acuteness of the inflammation, and in distribution according to its seat. Although, as a rule, pain is considerable, other signs of irritation, stiffness and muscular spasm, are generally inconspicuous, and the symptoms resemble, and are merged in, those due to the disease by which the meningitis is produced.

Diagnosis.—The most important indications of the chronic form are the vertebral pain and the radiating pains, with other symptoms of irritation of the nerve-roots, combined with the signs of pressure on the cord. These symptoms are seldom distinct from those of the cause of the external inflammation; the meningitis is indeed the chief mechanism by which the local disease causes its nerve manifestations.

The acute form causes symptoms that have much in common with those of internal meningitis. It is indeed doubtful whether the diagnosis can be made from the character of the nerve symptoms alone. The special indication of the seat of the process is furnished by evidence of its extension to or from the structures outside the spine. If such acute symptoms are secondary to external disease that clearly preceded the meningeal inflammation, this may safely be assumed to be outside the dura mater. In a case of apparently primary meningitis a careful watch should be kept on the tissues of the back; any sign of deep œdema among the muscles beside the vertebral column, in such a case, is probable evidence of commencing purulent inflammation extending from within, and the development of acute local inflammation in either the pleura, posterior mediastinum, back of the abdomen, or behind the pharynx, has the same significance. It is important that the nature of such symptoms should be kept in mind, as otherwise the occurrence increases the perplexing character of the case. It is probable that the nature of many cases, which remain undecided during life, would be rendered clear by repeated careful observation.

Prognosis.—The acute affection is exceedingly grave. Recorded cases have ended in death, but this termination has been a condition of the diagnosis; and it is possible that some cases which have recovered, in which the exact seat of the inflammation was uncertain, may have been instances of this form, and that the fatality of the disease may not be so great as published facts suggest. But on this point only future observations can throw light. Chronic external meningitis which results from caries is only serious in the compression it exerts on the cord.

puncture of the sac in spina bifida. (2) The exposure of the back to cold has been the apparent cause in rare cases; and still more rarely the prolonged exposure of the spine to the sun, "spinal insolation," has seemed to be effective. (3) Adjacent inflammation has sometimes caused internal meningitis, but in such cases external meningitis has of necessity been first produced, and the inflammation has passed through the dura mater. Hence all the causes of acute external meningitis are also occasional causes of the internal form. But, as already stated, such extension through the sheath is very rare, and there is some doubt as to the precise nature of many of the cases in which it has been supposed to occur. We have seen that in many of the instances in which external meningitis has been ascribed to adjacent suppuration, the latter has been secondary, and the meningitis has been the result of a primary blood-state. When in such cases there has been both internal and external meningitis, it is probable that the former has not been due to actual extension through the dura mater, but that both have been the simultaneous result of a general cause.

(B) In a few cases acute internal meningitis is due to the extension of inflammation from the cerebral membranes. Such extension is rarely traced, however, except in two classes of cases:—(1) Those in which slight meningitis is found in the upper cervical region, as far as gravitation favours the descent of solid particles in the cerebro-spinal fluid as the patient lies in bed. The inflammation ceases when the spine becomes horizontal: for the most part symptoms are slight or absent in such cases. (2) In cases in which a general spinal meningitis has been secondary in point of time to inflammation of the cerebral membranes the affection has generally been due to some cause capable of acting upon both, such as the local presence of tubercles or a morbid blood-state of the character to be immediately mentioned.

(C) The most acute and severe forms of internal meningitis are due to a morbid blood-state, either septicæmia or a virus allied to that which causes the cerebro-spinal form. Purulent meningitis occurs in cases of surgical and puerperal septicæmia, both with and apart from a similar affection of the cerebral membranes. Acute inflammation, purulent when intense and prolonged, occurs also as an isolated malady, sometimes without apparent exciting cause, sometimes after exposure to cold. In such cases organisms have been found in the spinal fluids, analogous to those of the cerebro-spinal form, in connection with which the pathological relations will be more fully considered. It probably results from a blood-state not far removed from those which cause some other forms of internal inflammation, and which give rise to acute articular rheumatism. General exposure to cold must be assumed to cause so special an effect only through the agency of toxæmia, such as that which has been already considered as underlying the rheumatic form of acute polyneuritis. Such exposure has been apparently rendered effective in some instances by coincident menstruation, which may have produced a special susceptibility to the attack, or favourable conditions for the development of some organised virus, such as seems to be the essential factor in the causation of these forms. The exposure to cold that has been effective has presented all the variations that are met with in the case of other diseases thus produced, variations which are common to so many maladies that they can have no special relation to any one of them. Lastly, as a crucial example of this origin of the disease, is the important fact that it may follow, or occur in the course of, certain acute specific diseases, as one consequence of the virus that produces them.

PATHOLOGICAL ANATOMY.—Internal meningitis is usually of wide extent, since the inflammation spreads readily in the loose tissue of the arachnoid. Probably also the movement of the cerebro-spinal fluid aids in the extension of local forms and in the descent of intracranial inflammation by conveying irritant material. In the case of a patient who had been in bed for some time the precise level at which such slight descending meningitis ceases may be that at which the vertebral column becomes horizontal. In the earliest stage the only change is congestion in the pia mater, which is reddened from vascularity, and may be dotted with ecchymoses. The inner surface of the dura mater, and the substance of the spinal cord, may be similarly congested. When the inflammation is further advanced, in the stage in which the condition most often comes under observation, the pia mater and arachnoid are opaque and thickened, and an "exudation" of inflammatory products, greyish yellow in tint, may cover the pia mater and occupy the meshes of the arachnoid, forming a layer over the cord. This exudation varies in its consistence, and may be semi-purulent in aspect; in the suppurative form the membranes are infiltrated with pus, which also covers their surface. The inner surface of the dura mater usually presents similar changes, and the inflammatory products may fill up the whole space between the dura and pia mater, thus connecting the two membranes and surrounding the nerve-roots (Fig. 94). The microscope shows abundant leucocytal elements, and the larger round and spindle cells that are common in all inflammatory products. The former corpuscles, which resemble, and are probably identical with pus-corpuscles, may be abundant, even when the exudation has not a distinctly purulent aspect; when it has, they constitute almost the whole of the material, lying among the fibres of the arachnoid (Fig. 95). The vessels are dilated, and their sheaths distended with cells. The spinal fluid is increased in quantity, and turbid from flocculi, or even purulent in aspect. The nerve-roots are covered with exudation, and are often swollen and reddened from invasion by the inflammation. But they do not always suffer, even in purulent meningitis, as Fig. 95 shows, in which a nerve-root is almost normal in aspect, and its sheath is unaffected, although it is surrounded by pus-cells. The spinal cord is often invaded by the inflammation; it is then reddened, pale, and softened, and the microscope shows the tissue changes common in other forms of myelitis. The change is always most marked in the periphery of the cord, and may occur in wedge-shaped areas, having the apex directed inwards, and coalescing at the surface. The peripheral arterioles come from the pia mater (see Vessels), and the continuation of their perivascular spaces with the lymphatic interstices of the membranes renders it easy to understand the invasion of the cord. Nevertheless in some cases, especially of purulent meningitis, the pia mater itself is little affected, even when the arachnoid is filled with pus, and in these cases the spinal cord may be normal, as in the example shown in Fig. 94. If recovery takes place, the inflammatory products may undergo cicatricial changes, the membranes remaining opaque and adherent, and there may be a permanent excess of arachnoid fluid. The changes in the cord may lead to sclerosis, widely spread, or limited to certain spots, and from these secondary degenerations, ascending and descending, may develop.

FIG. 94.

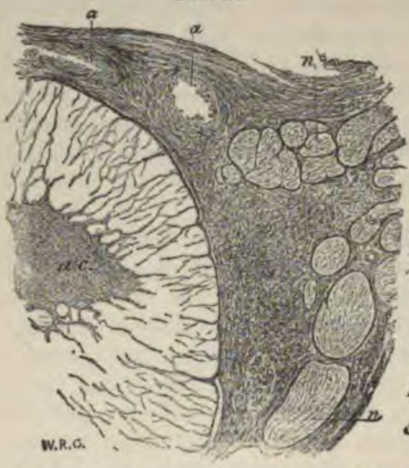

FIG. 95.

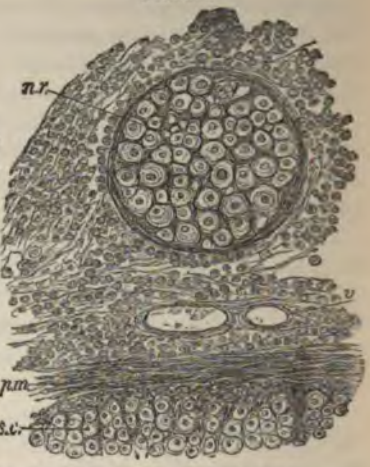

FIG. 94.—Purulent meningitis; portion of spinal cord and membranes; the space between the pia mater and dura mater is occupied by inflammatory products, pus, &c., in which the nerve-roots, *n n*, are embedded. *a a*. Cavities which had apparently been filled with liquid pus. From a case of septic origin, secondary to caries of the jaw.

FIG. 95.—From the same; meshes of arachnoid infiltrated with pus-corpuscles. A nerve-root, *n. r.*, although surrounded by pus, is perfectly normal; and so also are the pia mater, *p. m.*, and the peripheral layer of the spinal cord, *s. c.*

The area affected varies in different cases; the membranes are involved in their whole extent in most acute forms. Usually the affection is greater on the posterior than on the anterior surface; probably on account of the influence of the recumbent posture, which determines the passage backwards of the lymphatic fluids containing material capable of exciting and increasing the inflammation.

In tubercular inflammation the amount of exudation is usually small, and it may be absent. It is often grey and gelatinous in appearance, and in it are scattered the greyish or whitish tubercular granulations. Similar granulations may usually be found in abundance upon the inner surface of the dura mater. Such grey granulations are often found upon the spinal membranes in cases of tuberculosis when there is no inflammation, even when the cerebral membranes are intensely inflamed. The arachnoid, especially that covering the cauda equina, may appear as if dusted over with grey particles, so abundant are the granulations.

In many cases the signs of spinal meningitis are associated with those of inflammation of the membranes of the brain, especially of those about the base and posterior part of the brain. The continuity of the spinal and cerebral inflammation may be obvious or indistinct. In the latter case the connecting inflammation has apparently been much slighter than that in the base of the brain or around the cord, so slight, indeed, as to have left no distinct traces, the inflammation having apparently extended in consequence of the passage of morbid material by the cerebro-spinal fluid. In cases of slight cerebral meningitis, in which the patient has been recumbent, signs of spinal meningitis, opacity of the arachnoid and its distension with slightly turbid fluid, may exist only in the cervical region, ceasing opposite the upper dorsal vertebræ, and thus extending as far as gravitation (the head being slightly raised on a pillow) favoured the descent of inflammatory products. But in the violent purulent cerebro-spinal meningitis, which is occasionally produced by a septic influence, pus around the cord is often continuous with that which bathes the base of the brain. In a case of this kind, in which the meningitis was secondary to acute double purulent otitis, Leyden found abundant active micrococci, very similar to those associated with erysipelas. In other acute cases organisms have been found similar to those of pneumonia, and met with also in the epidemic cerebro-spinal form.

Symptoms.—Slight pain in the back, and malaise, may precede the acute onset of the symptoms. This is usually marked by a rigor, by pyrexia, and by severe pain in the back. The latter varies in position according to the locality of the inflammation, but is often felt along the whole extent of the spine. Pain also radiates along the distribution of the nerves, round the trunk, or to the extremities. This excentric pain is paroxysmal and intense; sharp, darting, burning, or constricting. The pain in the back is usually constant, with exacerbations. It is often increased by movement, usually also by pressure, and by the application of a hot sponge to the skin. It is no doubt due to the irritation of the inflamed meninges, while the radiating pain is produced by the irritation of the sensory nerve-roots.

Muscular spasm usually comes on at the same time as the pain. It shows itself first in rigidity of the muscles of the back, most marked, if the inflammation is local, in the neighbourhood of the inflamed part. This rigidity is an important and characteristic symptom. It is often first observed in the neck, probably on account of the mobility of the part. When slight it may merely cause retraction of the head, or stiffness of the back, or may be so general and so severe as to cause opisthotonos, resembling that of tetanus. The spasm usually involves also other trunk muscles, especially those of the abdomen, which become hard and cramped. The limbs also become rigid, and painful cramp-like spasms occur in them, especially on attempts to move. The spasm is probably partly due to the irritation of the motor nerve-roots, and is partly reflex, from the irritation of the sensory roots and the nerves of the pia mater. There is usually great hyperæsthesia of the skin to all forms of stimulation, and also increased sensitiveness of the muscles, especially in the lower limbs. Pressure on the arms may cause no uneasiness, while a similar pressure on the legs occasions great pain. Reflex action is usually increased at the beginning. Constipation is common, and so is retention of urine, in spite of irritable attempts to empty the bladder—the result, apparently, of spasm of its muscles, including the sphincter, which resists the action of the detrusor. Dyspnœa may result from the spasm of the thoracic muscles, and may be almost suffocating in its severity. The pulse may be frequent or retarded. The temperature is raised, sometimes, however, to only a slight degree. Cerebral symptoms, headache, delirium, coma, occur when inflammation has extended within the skull. The "Cheyne-Stokes breathing" may be present, from implication of the medulla. The function of the vaso-motor nerves (which leave the cord by many of the anterior roots) is also deranged; the dilatation of the vessels, that follows a stroke on the skin of the trunk, is excessive and prolonged (meningeal streak, *tache spinale*).

As the disease progresses, the symptoms of irritation give place to those of paralysis, which may be most marked where the rigidity was greatest. The limbs become relaxed, and feeble or powerless. Sensibility becomes lessened or lost. Reflex action disappears, the muscular power in the limbs becomes so reduced that the patient is scarcely able to move; the heart shares the universal prostration, and death may occur from asthenia, or from paralysis of the respiratory muscles. Towards the end there is sometimes considerable rise of temperature. In some cases the symptoms become less progressive and the disease passes into a less acute stage; the pains persist, the loss of power continues and may even slowly increase. Death may occur, after weeks of suffering, from the effects of bedsores or from secondary kidney disease, due to the retention of urine, and facilitated by trophic derangement. On the other hand, in slight cases, the signs of irritation may lessen and pass away, while those of structural damage, paralysis and anæsthesia, may remain, and to these may be added muscular atrophy and contractures, from the secondary consequences of the lesions of the nerve-roots. Such persistent symptoms vary much in extent and degree, according to the position and intensity of the morbid process. Ultimately the symptoms of damage to the cord may either slowly increase, in consequence of the spread of chronic myelitis set up by the acute mischief, or, on the other hand, the symptoms of the meningitis may pass entirely away.

The symptoms above described vary in their distribution, according to the position of the disease. When the membranes over the lumbar enlargement are chiefly affected, the pains, hyperæsthesia, and cramps are confined to the legs and loins. When the disease is in the dorsal region there may be similar hyperæsthesia and spasm in the legs, but the pain and cramp extend higher, and involve the trunk. If the cervical region is affected, the symptoms extend to the upper extremities, the dyspnœa may be great, and there is often difficulty in swallowing. The action of the heart is sometimes deranged, and contraction or dilatation of one or both pupils may occur. Extension to the brain is marked by vomiting, general headache, delirium, and paralysis of cranial nerves, of which the first to suffer are the spinal accessory and hypoglossal. If such symptoms preceded those of spinal meningitis, we may conclude that the inflammation commenced within the skull.

The symptoms vary somewhat according to the nature of the inflammation. In purulent meningitis, as already pointed out, the symptoms of irritation are sometimes very slight, apparently because there is little tendency to invade the nerve-roots. In the case from which Figs. 94 and 95 were taken there were hardly any symptoms to suggest meningitis, and although there was paraplegia, it was probably produced by the mere pressure of a large collection of pus on the spinal cord; there were no pains or spasm. The case was one of septic origin. It was apparently a pure arachnitis, and the tissue of the pia mater was able to resist invasion so completely as to protect the structures beneath it even from disturbance of function.

The duration of the acute symptoms varies from a day or two, in severe cases, which end in death, to two or three weeks, in cases of less severity, which may end in either death or recovery. The duration of the subacute and chronic symptoms that supervene is to be measured by months, and sometimes by years.

The symptoms of tubercular spinal meningitis resemble those which have been described, but are usually less intense. There is pain in the back and loins, with stiffness of the spine and retraction at the neck, so that it may be difficult to bend the head forwards. There are also variable rigidity, hyperæsthesia, and tingling in the limbs, followed by lessened sensibility and paraplegic weakness. These symptoms occasionally come on in the later stage of cerebral tubercular meningitis.

Diagnosis.—The diagnosis of the disease rests on the pain in the back, the retraction and rigidity of the neck and spine, the hyperæsthesia and spasm in the limbs, excited especially by attempts to move them, on the acute onset of the symptoms, and on their association with pyrexia. This grouping of the symptoms is sufficiently characteristic, as is shown by the fact that it is extremely rare for them to be simulated, even remotely. The chief difficulty in actually acute cases is presented by the forms that run an almost latent course, especially by the secondary purulent form, which has so little tendency to invade the nerve structures, and a correspondingly slight tendency to derange their functions, and even to irritate them. In pure *myelitis*, on the other hand, pain in the back is absent or trifling; paralysis occurs early and is the leading symptom, and there is little or no spasm in the limbs in the early stage of the affection. Often, however, some meningitis occurs at the onset of acute myelitis, and then some pain in the back and slight rigidity in the limbs may precede or accompany the onset of the paralysis. In such cases the predominance of the meningeal or cord symptoms must determine the category in which the case is to be placed. *Meningeal hæmorrhage* most nearly resembles meningitis in its manifestations, and does so necessarily, because it produces inflammation; the distinction chiefly depends on the onset, and will be considered in the next section. *Hæmorrhage into the spinal cord* can scarcely be confused with inflammation of the membranes, because the only common symptom, pain in the back, is confined to a definite spot.

Tetanus is attended by rigidity of the back, and by spasm, and presents a closer initial resemblance to meningitis than most other affections with which it may be confounded; but there is no fever at the onset, trismus is an early and obtrusive symptom, and the paroxysms of muscular spasm are excited by peripheral impressions much more readily than in meningitis, in which they occur chiefly on attempts to move. Only the "rheumatic" form of tetanus would give rise to difficulty; the onset of the traumatic form after an injury should prevent any doubt. *Rheumatism of the muscles* may cause pain in the back on movement, and the resulting rigidity may prevent movement; but there is not the spontaneous pain which attends meningitis, nor is there distant spasm. Some difficulty may be caused by rheumatism of the cervical muscles in young children; this may lead to retraction of the head, and if it comes on acutely, after exposure to cold, considerable doubt as to its nature may at first be felt. But it remains stationary, and the freedom of the patient from spontaneous or radiating pains, and from any affection of the nerves, soon enables a reassuring opinion to be formed.

The diagnosis of the form of meningitis depends on the recognition of the cause of the inflammation. When spontaneous and acute the case is probably of the "primary" form, which depends on some toxic agency, probably often an organised virus, and is really a partial "epidemic" form. If it occurs under the conditions that give rise to surgical septicæmia, or after childbirth, purulent meningitis is almost certain, and the inflammation may be far more intense than the severity of the symptoms suggest. The diagnosis of tubercular spinal meningitis depends on the combination with cerebral meningitis, which usually precedes the spinal symptoms, and on the gradual and insidious onset. Indications of the tubercular or scrofulous diathesis are commonly present in the state of other organs, or to be ascertained from the family history of the patient.

PROGNOSIS.—The prognosis is grave in all cases. It is worse the more severe and acute the symptoms, the higher the temperature, and the sooner the symptoms of irritation give place to those of paralysis. It is worse also when the disease is due to serious lesions of the spine or to tuberculosis, than when due to cold, and worse in the so-called "spontaneous" cases than in those which result from traumatic causes. Recovery is more probable in middle life than in early or advanced age. The previous health of the patient also influences the prognosis. It must always be remembered that even if the patient survives the period of acute inflammation, serious permanent damage may remain.

TREATMENT.—Perfect rest and quiet are of the greatest importance throughout the course of the disease. All sounds should be, as far as possible, excluded; the light should be subdued, and all bodily movement and mental exertion as far as possible avoided. The vascular disturbance of inflammation is intensified by all functional excitement of the too irritable structures, and continuous freedom from such excitement, as far as it is possible to obtain the freedom, is of paramount importance. Every attack of spasm means greater vascular disturbance, and the circulatory derangement entails leucocytal infiltration and other structural consequences.

Although it is undesirable that the spine should be the lowest part of the body, yet, in acute meningitis, it is scarcely possible for the patient to lie in any other posture. The prone position interferes with respiration, and both it and a lateral posture entail, directly or indirectly, greater muscular exertion, and hence more frequent and more severe attacks of spasm, with secondary harm out of proportion to that which the posture could prevent. Here, as so often in therapeutics, skill is shown and success secured, if it is within reach, by knowing when to permit no compromise, and when to adopt one. The dorsal position must be permitted if in it the spasm is much less than in others. Dry, or, in robust patients, wet cupping, or leeching, along the spine may be employed at the onset, and be followed by the local application of cold in traumatic or hæmorrhagic cases, and of heat in others—the principles being the same as in myelitis. Counter-irritation, by blisters or repeated sinapisms, is more useful when the disease is subsiding than at the onset. In cases that are due to cold, free diaphoresis often does good; a hot air or vapour bath should be employed at the onset of the treatment. A warm bath may be followed by moist packing for several hours. The relief thus given is sometimes very great. The bowels should also be freely opened.

The only internal remedy which has been held, for long, in general repute, as capable of influencing the inflammatory process, is mercury. The confidence placed in it of old is not altogether unwarranted. It should be given until there is a slight affection of the gums (the only evidence that enough is in the system to act on the tissues), and inunction is unquestionably the best way of administration. We know that it must then enter the blood before it can escape by the bowel; we can regulate its dose as we need, for we estimate it by the influence on the gums; and by rubbing it in over the part affected we can combine some counter-irritant influence (as by an irritating agent added to the ointment used), and we necessarily secure the simultaneous action of the two agents, for the largest quantity of mercury in the blood will coincide with the most considerable reflex action on the vessels. Lastly, the disturbance of the stomach and bowels is certainly less than when their mucous membrane is chosen as the path into the body. The oleate of mercury may be rubbed in along the spine. Iodide of potassium seems to have little influence over acute inflammation.

It is necessary to give sedatives for the relief of the pain and spasm, if the application of cold does not suffice. Of these, morphia, given by the skin or the mouth, is the most effective, but vomiting from its use is a grave drawback; fortunately it does not readily occur under the circumstances. Sometimes inhalations of chloroform are necessary to relieve the suffering, and may be even more effective than morphia. In slight cases relief may be afforded by belladonna, or by atropia injected beneath the skin. Chloral, or chloral and bromide, or any of the milder sedatives, may be given if there is insomnia. Bromide alone, unfortunately, has very little power to lessen the spinal reflex action. Frequently, the spinal ice-bag is the most effective and least injurious.

When the disease has passed into a chronic stage, iodides have been thought useful. Counter-irritation may be employed freely with advantage. Tonics, iron, quinine, and even strychnia are beneficial. Warm baths, as those of Bath and Aix-les-Bains, seem sometimes of service, especially employed as hot douches to the spine. But the acute form is followed by a tendency to the slow subsidence of its effects, which makes the influence of such measures difficult to estimate. The local consequences, muscular atrophy, contracture, &c., need special local treatment by electricity, rubbing, and the like.

Epidemic cerebro-spinal meningitis is described in the section on diseases of the cerebral membranes (Vol. II).

CHRONIC INTERNAL MENINGITIS.

Chronic inflammation of the membranes of the cord, within the dura-matral sheath, is divided into two forms, according as it begins in, and chiefly affects, the dura mater, *chronic internal pachymeningitis*, or the pia mater and arachnoid, *chronic leptomeningitis*. Although these forms sometimes present distinct clinical and pathological features, they have, when of considerable degree, many characters in common; they own the same causes, and need the same treatment. Hence it is most convenient to describe them together as forms of internal meningitis. The condition termed *hæmatoma of the dura mater* depends on a form of hæmorrhagic inflammation.

Chronic meningitis is seldom fatal, and is rare as a general and primary malady. As such, therefore, our knowledge of it is still scanty, and the opinions formerly current have had to be largely curtailed. It was inferred that chronic symptoms, analogous to those which, when acute, are due to acute meningitis, were evidence of a chronic form. This was, therefore, assumed to be the cause of a group of symptoms, of which spasm is the prominent and dominant feature, which are now known to be due solely to a morbid state of the spinal cord itself, and to be consistent with a perfectly normal state of the membranes. Hence "chronic meningitis," as it was recognised twenty years ago, as a "clinical entity," has ceased to exist, or rather has passed into the pathological conception of "primary lateral sclerosis." The definite knowledge we now have of general primary chronic meningitis is limited. The important fact regarding it is that its symptoms differ from those of the acute form more widely, perhaps, than do those of any other chronic inflammation. Yet this difference depends on a comparatively small element—on the fact that only acute inflammation causes acute irritation of the motor and reflex structures. The chronic form may irritate the sensory nerve-roots, but its motor manifestations are chiefly the result of pressure, and even to this much of the pain may be due. The pressure may cause spasm, but it is a slow tonic contraction, wholly unlike that of the acute form. The difference in the effect of the two forms of inflammation suggests many problems that deserve investigation, but their consideration would be out of place here.

Causes.—Chronic internal meningitis, in every form, is most frequent in adult age, and, like acute inflammation, affects men more frequently than women. In its general and primary form, it has been thought to occur more readily in persons with neurotic heredity, although the influence of this is doubtful. Debilitating influences of various kinds predispose to the disease, and prolonged over-exertion has been thought sometimes to produce it. Among exciting causes the most important is severe and repeated exposure to cold. Traumatic lesions, concussion, &c., are occasional causes. It may result by extension from inflammation outside the dura mater and from chronic inflammation of the substance of the cord itself. But their influence in producing chronic internal inflammation (except as a sequel to the acute form) is a subject on which the opinions of the past have still to be subjected to careful comparison with ascertained facts. It is probable, however, that the condition does sometimes follow concussion of the spine that has no immediate or acute consequences, and the chronic stage into which an acute traumatic meningitis subsides may last so long as to throw its initial form into the shade, and may not only persist with independent pertinacity, but may extend without relation to the original seat. Thus we must distinguish as effects of injury, the local and the general forms. The latter are predisposed to by the influences that seem to facilitate the occurrence of the primary form, and in these cases the influence of the injury often seems to be trifling, and the existence of the morbid state is not always beyond doubt.

Local chronic meningitis may also result from any chronic disease either of the membranes themselves, the bones, or the spinal cord. These need not be enumerated in detail. It occurs in all cases of compression, and especially in all forms of chronic myelitis that involve the superficial layers of the cord.

Lastly, certain general morbid states are frequent causes. The first is alcoholism, which may give rise to general inflammation, involving both the pia arachnoid and the superficial layers of the cord in various degrees. A second and also common cause is syphilis, which may produce either pachymeningitis, surrounding the cord in the way presently to be described, or local leptomeningitis, irregular in position and in effects. Lastly, in very rare cases tubercle has given rise to a chronic inflammation, chiefly of the inner surface of the dura mater. Hæmorrhagic pachymeningitis occurs especially in the insane, but has been met with as a consequence of chronic alcoholism and after injuries.

PATHOLOGICAL ANATOMY.—In slight and moderate degree, there is merely opacity and thickening of the membranes affected, sometimes with distension of vessels or minute spots of extravasation. The opacity of the arachnoid may be such that the spinal cord cannot be seen through it. The inner surface of the dura mater may be granular when it is not otherwise changed. The spinal fluid is increased in quantity and is turbid. When the changes are greater in degree, the dura mater and pia mater may be connected together by a layer of inflammatory tissue of considerable thickness, so that it may be impossible to say in which membrane the disease commenced. The microscope shows the ordinary elements which result from inflammation, cells of various kinds, many lymphoid and pus-like corpuscles, and distended vessels, often incrusted by similar cells. Frequently also the pia mater is transformed into a thick irregular layer of homogeneous tissue in which no distinct cell-elements can be perceived, and only faint indications of a fibrous structure (Fig. 96). The walls of its vessels may be greatly thickened by similar material.

The nerve-roots passing through the diseased membranes very seldom escape, as they may in the acute form, but are inflamed, reddened, and swollen in the active stage, and afterwards compressed and atrophied, if the amount of new tissue formed about them is considerable. The fibres suffer especially when the inflammation involves the dura mater, on account of the unyielding character of the fibrous sheaths which the nerves receive from that membrane. When the pia mater is thickened, scattered tracts of connective tissue may be seen in the substance of the nerve-roots, between the fibres (Fig. 96, *t*).

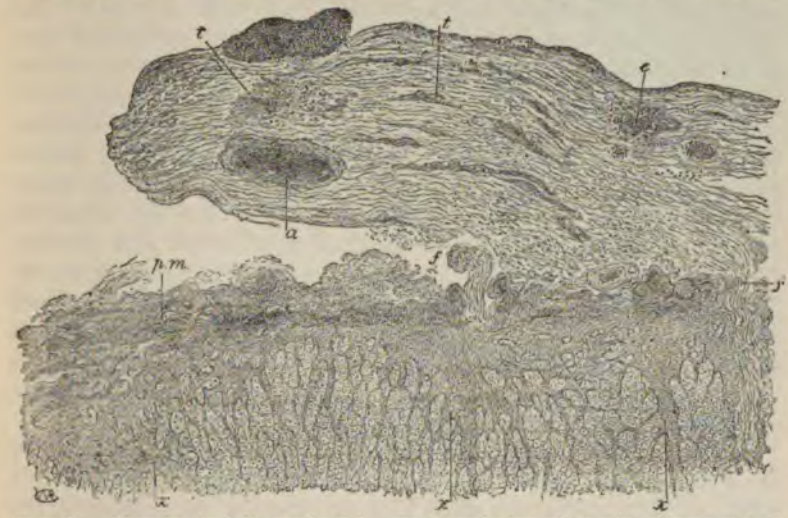

FIG. 96.—Chronic alcoholic meningitis. Section of edge of anterior column and of a large nerve-root; carmine preparation. *p.m.*, pia mater irregularly thickened and transformed into amorphous-looking tissue, from which wedge-shaped branching tracts (*x x*) extend into the white substance; *f.f.*, fasciculi of nerve-fibres entering the cord; *a.*, an artery in the nerve-root, enlarged and with thickened walls; *t. t.*, tracts of amorphous connective tissue; *e.*, a small extravasation.

The spinal cord presents the same variations in the degree in which it suffers. It may be little affected, but is frequently damaged by the extension to it of the inflammation of the pia mater, which causes softening, vascularity, loss of distinction between grey and white substance, breaking down of nerve-elements, and infiltration by lymphoid and other inflammatory cells. Ultimately indurating tissue remains, constituting an irregular zone of peripheral sclerosis. From the thickened pia mater tracts of similar tissue may extend into the cord, becoming narrower as they pass inwards and sending out branching trabeculæ (Fig. 96, *x*). Between these tracts the nerve-fibres are more or less damaged, partly by the inflammation, and partly by the compression produced by the newly-formed tissue.

This peripheral sclerosis associated with chronic meningitis, especially in the neighbourhood of injuries, frequently damages tracts that undergo secondary ascending degeneration—postero-median column, direct cerebellar tract, and ascending antero-lateral tract. The result is to produce a mixed lesion, the precise nature of which is not always easily distinguished and has certainly often been mistaken. The equivocal aspect of the process is probably increased by the fact that, as we have seen, secondary degeneration entails an interstitial process that is prone to assume an inflammatory character, especially when set up by an irritative lesion. Hence the peripheral sclerosis set up by the meningitis is apt to carry with it a certain amount of inflammation in the pia mater, far beyond the region to which this would otherwise be limited. A similar thickening of the pia mater is, however, met with over areas of the cord that are the seat of other forms of sclerosis, both those that are systemic, as in tabes, and those that are random, as in the insular form. It is probably secondary in all cases, but it formerly gave rise to some erroneous conceptions of the nature of such degenerations (see Locomotor Ataxy). A still more considerable implication of the pia mater is met with over regions that are the seat of focal chronic myelitis.

In the ordinary form of chronic meningitis, the membrane in which the inflammation begins may be much more affected than the other, although the latter is very rarely healthy, except in the slightest cases. Inflammation beginning in the pia mater and arachnoid is often extensive in range, and may be considerable or slight in degree. That which commences on the inner surface of the dura mater is frequently limited in extent, but is accompanied by the formation of a large amount of new tissue, and hence has been termed *hypertrophic internal pachymeningitis*. It constitutes a very important variety, giving rise to peculiar and grave symptoms which were first carefully studied by Charcot and Joffroy. It affects most commonly the cervical region, but sometimes occurs at the lower part of the cord. On opening the spinal canal a fusiform tumour is seen, the outer surface of which is the unaltered outer surface of the dura mater, and on section the enlargement is found to depend chiefly on a great thickening of the inner part of this membrane, sometimes amounting to a quarter of an inch; several layers of new tissue can often be distinguished. The pia mater may be normal, but is more commonly thickened, and it is often united by the thickened arachnoid to the tissue proceeding from the dura mater. The cord is compressed and commonly softened at the spot, and it presents signs of inflammation. The nerve-roots are also damaged and compressed by the newly-formed tissue. Sometimes at the spot most affected, the cord may be surrounded by a ring of new tissue of cartilaginous hardness. In other cases the thickening of the dura mater is slighter and more diffuse, affecting occasionally a wide extent of the membrane. In rare cases the membranes about the cauda equina may alone be affected—the dura and pia mater adherent, and the nerves united in a fibrous mass.

The white fibroid or cartilaginous plates found so often in the arachnoid after death are probably not connected with preceding inflammation. In most cases in which they are found, no symptoms have existed during life. It is said, however, that when they are numerous and extensive they may give rise to symptoms closely resembling those of chronic meningitis (Vulpian). But fibroid plates in the dura mater have been seen in a case in which previous symptoms suggested that they resulted from chronic inflammation (Jaccoud).

The rare condition termed *internal hæmorrhagic pachymeningitis*, or *hæmatoma of the spinal dura mater*, is similar to, and commonly associated with, that which affects the cranial membrane, and will be afterwards described. In this, a reddish-brown exudation covers the surface of the membrane, and is composed of fibrin and extravasated blood; the latter may be encysted in small cavities, or may be in various stages of transformation. The change commonly extends over a great part of the dura mater, and is apparently the result of hæmorrhage into inflammatory tissue.

The local form of syphilitic meningitis presents an irregular thickening of the pia mater and arachnoid, with the extensive production of new tissue in the affected region characteristic of this variety, so that it often resembles a diffuse growth as much as a chronic inflammation. The new tissue has a peculiar gluey aspect, and presents a tendency to caseous degeneration and fibroid change, so that scattered or coalescing cheesy spots are ultimately met with in tracts of mixed fibrous and gelatinous aspect, the latter appearance predominating in the more recent parts. This process of growth may attain such a development at some spot as to constitute a distinct "gumma," which may invade the substance of the cord, growing inwards from the pia mater. A similar process of cell-growth may be sometimes traced with the microscope into the substance of the cord, along the tracts of neuroglial tissue and peri-arterial spaces, when there is no invasion distinct to the naked eye.* A more limited affection of the walls of the arteries of the pia mater, extending into the cord, has also been thought to be syphilitic in nature; the whole thickness of the wall of the arterioles may be the seat of inflammatory thickening, in consequence of which the calibre of the vessels is narrowed.† There is, however, some reason to think that a similar affection of the vessels may occur apart from syphilis.‡

In the very rare chronic tubercular meningitis the inner surface of the dura mater is covered with a layer or layers of tubercles, in various stages of development, mingled with inflammatory products. The arachnoid may also contain degenerating tubercles, and the two membranes may be united together by the new formation. The condition occurs independently of bone disease.§

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* Gilbert and Lion, 'Arch. gén.,' 1889.

† Schmaus, 'Deut. Arch. f. kl. Med.,' 1889.

‡ Hochhaus, 'Thesis,' Kiel, 1889.

§ Weiss, 'Wiener med. Wochensch.,' 1885.

Symptoms.—The symptoms of chronic internal meningitis, like those of the acute form, are due to the irritation of the membranes, to the damage to the nerve-roots, and to that of the cord. But the absence of acuteness in the process involves, as we have seen, a difference which produces a contrast, rather than a resemblance, in the manifestations of the two diseases; the spasm which dominates the aspect of acute meningitis is almost entirely absent in the chronic form. When this is eliminated, similar disturbances of function remain, but their relative subordination renders the similarity in the features of the two maladies unobtrusive. Still, the three pathological elements just mentioned occur in the chronic as well as in the acute form, and in different cases one or another of the corresponding sets of symptoms predominates. Hence the varieties, while possessing many characters in common, may differ considerably in their clinical aspect. In some cases, we can infer with accuracy the special seat of the disease, but in many others we cannot carry our diagnosis beyond the conclusion that chronic internal meningitis exists, and are unable to say, even with probability, which membrane is primarily affected.

The common symptoms are these: pain in the back, increased by movement,—sometimes, however, amounting only to heavy dull discomfort,—and accompanied by some stiffness of the back, and, when in the cervical region, by some retraction of the head. The pain is increased by pressure on the vertebral spine, and its increase by movement may cause a fixation of mobile parts, as the neck, in some abnormal position. The pain is apparently due to the irritation of the meningeal nerves, and the rigidity is to be regarded as a reflex effect of this irritation. But these are not the paroxysms of spasm, so conspicuous and so distressing in the acute form.

More obtrusive, in most cases, are the radiating or excentric pains due to the irritation of the nerve-roots, and referred to the region in which the nerves are distributed,—back of the head, neck, arms, thorax, abdomen, loins, or legs, according to the position of the disease. These pains are often very severe, sharp, darting, burning, or rheumatoid in character, paroxysmal in occurrence, and sometimes worse at night. They may be accompanied by a painful sense of constriction, and by various unpleasant sensations—numbness, tingling, formication,—in the same areas. Hyperæsthesia may also exist, so that pain, sometimes thrilling in character, is produced by touching the skin, or by slight degrees of heat or cold, and a hot or cold sponge passed along the skin of the back may reveal the chief seat of the disease by the change of sensation at its level, as in the acute form. The hyperæsthesia may be more marked to one form of sensation than to another. Cutaneous eruptions, such as result from other nerve lesions, have also been observed in association with the pains. In the same regions, muscular twitching, tremor, or even spasm, may result from the irritation of the motor root-fibres. These symptoms correspond in their level to the part of the membranes chiefly diseased. When the lumbar membranes are affected, the radiating pains are felt in the legs; but when the disease is higher up, the legs may merely feel heavy, and be the seat of slight abnormal sensations.

After a time, weeks or months, the pains persisting or ceasing, certain paralytic symptoms manifest themselves in the regions in which the severe pains were felt, and are dependent on further damage to the nerve-roots. Sensation becomes lessened, or even lost, in certain areas, to touch, or pain, or both. The muscles in the same region become weak, and waste; usually with the reaction of degeneration for a time, and ultimate loss of all irritability. The wasting often affects the muscles irregularly, from the unequal damage to the roots and the fibres of single roots: entire groups sometimes atrophy from the complete interruption of the roots of one or more segments. Reflex action in these parts is lost. In the trunk this loss of reflex action, with anæsthesia, may be the chief local symptom, and is of much diagnostic importance. As the disease progresses, the cord itself suffers, by compression, or by extension to it of the inflammation. The parts below the seat of the disease then become paralysed, the legs are weak and the seat of dull heavy pain; reflex action in them may be increased, while their muscular nutrition continues good, unless the lumbar membranes are diseased, or the myelitis descends the cord. Defect of co-ordination in the legs is not uncommon, and is probably due to the implication of the fibres from the muscles in the posterior columns and cerebellar tracts. Power over the sphincters may be lost and bedsores form, but chiefly in cases in which the disease is severe over the lumbar region or cauda equina.

When the inflammation affects chiefly the pia mater and arachnoid (*chronic leptomeningitis*) the symptoms of meningeal irritation are conspicuous,—vertebral pain and rigidity of the back, with cutaneous hyperæsthesia, and the radiating pains may also be considerable. These symptoms are especially marked in cases of subchronic meningitis, such as are sometimes due to alcoholism. The local paralytic symptoms—anæsthesia and muscular wasting—may be absent in these cases, or may only come on in slight degree after the other symptoms have lasted for a considerable time. On the other hand, weakness and pains in the legs, from affection of the cord itself, may be early symptoms.

When the inflammation begins in the dura mater (*internal pachymeningitis*) the symptoms of spinal irritation, pain and stiffness of the back, although occasionally present, are much less prominent symptoms than are those which depend on the irritation of, and damage to, the nerve-roots. The radiating pains are very severe, and are often the earliest symptom. At a later stage the muscular weakness, followed or accompanied by wasting, chiefly attracts attention, and the wasting may be so great that the disease is mistaken for progressive muscular atrophy. This is especially the case when the lesion is in the cervical region, the "cervical hypertrophic pachymeningitis" of Charcot and Joffroy. In this affection, pains in the back of the head, neck, shoulders, and arms, often accompanied with a painful sense of constriction, precede the muscular atrophy. The wasting may involve many muscles, but usually those supplied by the radial nerve are less affected than the others; the escape of the long extensors of the wrist and fingers, while the flexors of the wrist and fingers and the interossei atrophy, leads to a peculiar deformity; the unopposed muscles cause persistent over-extension of the wrist; the phalangeal joints are flexed and the metacarpo-phalangeal joints are extended, but from the position of the wrist they are not over-extended, and the hand thus differs from the claw-like hand (p. 43). With this muscular wasting in the arms there is paraplegic weakness from the pressure on the cord. In rare cases the disease affects the membranes over the lumbar enlargement or cauda equina, and then the legs are the seat of the pains, paralysis, and atrophy already described, and the sphincters are early paralysed.

If the pachymeningitis is confined to the dorsal region, the root-symptoms are limited to the trunk. In such a case anæsthesia over the region supplied by the dorsal nerves was associated with paraplegia from compression of the cord. In another, areas of anæsthesia, with severe pains, existed over the legs, trunk, neck, and back of the head, without any indication of damage to the cord itself. These examples illustrate the great variations presented by different cases, owing to the different distribution of the disease. An important characteristic of this form is the fact that it involves the membranes equally around the cord, and so gives rise to bilateral symptoms.

Chronic syphilitic meningitis may cause symptoms resembling those of any other variety of chronic inflammation, even the hypertrophic pachymeningitis being sometimes due to this cause. But its most frequent and special characteristic is the focal character of the symptoms, and their tendency to be associated with those of partial and local damage to the spinal cord itself. This is in consequence of the tendency of local tissue-formation to be associated with the inflammation. Hence, pains of irregular distribution on one side are usually soon followed by impairment of sensibility, and often by weakness in the corresponding leg.

In hæmorrhagic pachymeningitis the symptoms are those of a slight chronic inflammation, without any distinctive features that can be referred to the hæmorrhagic infiltration.

DIAGNOSIS.—The diseases with which chronic internal meningitis is liable to be confounded, are different according as the symptoms of irritation, of damage to the cord, or of compression of the nerve-roots, predominate. By far the most important diagnostic symptoms are those that depend on the damage to the nerve-roots—their early irritation and later structural damage.

The spinal pain and stiffness may be mistaken for simple rachialgia or "spinal irritation," but in this condition the whole spine is tender, several separate points are much more tender than the rest, and there are not the radiating pains or paralytic symptoms of meningitis. Pain in the back is related in greater degree to posture and exertion, and it has a tendency to pass from the neck to the occiput. The patients have usually suffered from neuralgia in other situations, which may alternate with that in the back.

Peripheral pains, especially about the trunk and legs and shoulders, are prominent symptoms in some cases of muscular rheumatism or rheumatic neuralgia. In these, however, there are not the pain and tenderness of the spinal column; and although there may be apparent increased sensitiveness in the region of the pains, it is of the deeper structures and not of the skin, while the relation of the pain to movement and muscular action is a conspicuous feature.

Radicular neuritis is probably associated in many cases with some affection of the adjacent membranes, but meningeal symptoms are altogether subordinate; the symptoms are confined to the region supplied by the nerve-roots, and spinal pain is absent.

The muscular wasting may be mistaken for atrophy due to degeneration or subacute inflammation of the anterior cornua of the cord (progressive muscular atrophy, subacute polio-myelitis). The cervical hypertrophic pachymeningitis bears special resemblance to this disease, since the wasting in the arms is often associated with paraplegic symptoms in the legs. The distinction is that the atrophy, in the meningeal form, is preceded by severe pains, and often accompanied by areas of hyperæsthesia or anæsthesia, and is random in distribution. In chronic affections of the anterior cornua both pains and anæsthesia are usually absent, and the wasting often affects the muscles in a certain order, which will be described in the account of the disease. Thus, in a man who was supposed to have cornual disease on account of wasting of some muscles of both legs, irregularly distributed, a history of severe sharp pains, and the discovery of a patch of anæsthesia on one leg, the presence of a burning pain round one side of the trunk, and the irregular distribution of the wasting, led to a diagnosis of chronic meningitis damaging the nerve-roots; there was a history of syphilis, and appropriate treatment completely removed the symptoms. The pains also constitute a distinction from another affection which presents somewhat similar symptoms—syringomyelia. Chronic disseminated myelitis may cause muscular wasting, but does not cause the pain in the back, or the rigidity of the spine, which occur in meningitis, and the paralysis from damage to the cord is the leading symptom. It must be remembered that the two conditions of meningitis and myelitis are often combined, and the fact has to be recognised in our diagnosis.

Some authorities believe that the symptoms of "ataxic paraplegia," which result from combined lateral and posterior sclerosis, are often produced by the agency of chronic meningitis, invading the peripheral layers of the cord. It is not probable, however, that this mechanism is operative in the cases in which considerable paraplegia shows that the lateral pyramidal tracts are extensively involved. The diagnostic problem is considered in the chapter on this disease.

When the meningitis is lumbar, the pains in the legs may resemble some of those that occur in locomotor ataxy, and the knee-jerk may be lost in each disease; but the loss in meningitis is associated with muscular weakness and wasting, there is not the degree of ataxy common in tabes, and true "lightning pains" are not met with in meningitis. The greatest resemblance to chronic meningitis is presented by some cases of tabes in which the cord disease reaches a greater degree in the dorsal than in the lumbar region. There are then pains and anæsthesia in the trunk, and the atrophy in the legs may be little marked. In meningitis, if the membranes over the lumbar enlargement are normal, the knee-jerk is preserved and often increased, while it is lost in tabes. The anæsthesia and loss of the trunk reflexes are more general and uniform in tabes than in meningitis.

Caries of the spinal bones produces many of its symptoms by the agency of chronic meningitis, which always results from it. Hence the diagnosis depends on the recognition of the presence of the disease of the bone, in addition to that of the membranes.

Multiple neuritis, in the paralytic variety, presents nerve-symptoms of the same character as those of chronic meningitis, but the special symmetry and peculiar distribution of the atrophic palsy, the prominence of limb-tenderness, and the absence of cord symptoms, preclude such similarity as can cause actual confusion between the two diseases.

PROGNOSIS.—The differences between the forms of chronic meningitis, and between the degrees of the same forms, are so vast as to render the prognosis of the disease a matter of individual consideration in every case, to be estimated independently, from the pathological characters. The severe degrees of the affection are attended with danger to life, and even the slighter forms may entail serious consequences, since many effects of the disease, especially the damage to the spinal cord itself, tend to increase by the myelitic tendency which is set up. The neuralgic pains, which result from the damage to the nerve-roots, are extremely obstinate. But in many cases the symptoms ultimately pass away—the irritation ceases and the compressed structures slowly regain their function. The prognosis is most favorable in the cases which result from injury and from syphilis. But in all cases, even those of syphilitic origin, in which there is reason to infer considerable formation of new tissue, the prognosis must be cautious, since the cicatricial contraction of this tissue may perpetuate the damage to cord and nerves which its pressure has produced. The prognosis is better, in other cases, in proportion to the general health of the patient, to the moderate degree of the mischief, and to the extent to which its causes are under control.

TREATMENT.—Rest is essential; in severe cases it should be absolute, and in every case movements which increase pain should be avoided. The pain is the expression of irritation, which tends to maintain the morbid process. Measures, previously ineffective, will often be followed by improvement if rest is added. Posture is also of great importance. The prone couch, impracticable in acute meningitis, can often be employed in chronic cases with advantage, to lessen mechanical congestion of the spine. Warm baths and diaphoretic baths have been recommended as useful, especially in subacute cases. Next in importance to rest and posture is counter-irritation. Repeated sinapisms or stimulating liniments may be used if mild counter-irritation only is required, but in most cases greater good will result from more energetic means—blisters or the actual cautery. The last is especially recommended by Joffroy for the hypertrophic pachymeningitis. It is probable that posture favours the beneficial influence of counter-irritation in a very important degree.

Sedatives are usually needed to relieve the pain—morphia, chloral, Indian hemp by the mouth, subcutaneous injections of morphia or cocaine, or sedative liniments of chloroform, belladonna, &c. In employing sedatives it must be remembered that they will usually be needed, in chronic cases, for many months, and after a time, by the artificial need they create, they may apparently keep up the pain. It is therefore well, if possible, to alternate two or more sedatives, to make cocaine take the place of morphia as much as possible, and to lessen the dose from time to time tentatively, and without the knowledge of the patient.

Iodide of potassium and mercury are the agents that have most influence over the morbid process itself. Both are effective in syphilitic cases, but in other forms of the disease mercury has far more influence than iodide; it may be rubbed in over the spine, as recommended in the acute form. The ointment and oleate may be rendered counter-irritant, if necessary, by the addition of cantharides ointment. In many cases, tonics, as iron, quinine, and cod-liver oil, are needed. The muscular wasting which results from damage to the nerve-roots requires treatment by galvanism, rubbing, and by passive movement, to prevent contractures. If hæmorrhagic pachymeningitis is suspected, ergotine may be used in addition to the measures suited for the ordinary form. Treatment appears, however, to exert little influence upon it.

Other measures are the same as have been described for the acute form.

HÆMORRHAGE INTO THE SPINAL MEMBRANES; HÆMATORACHIS.

Spinal hæmorrhage may be outside the dura mater, between it and the bones (*extra-meningeal*), or within the dura mater (*intra-meningeal*). In the latter situation the extravasation may be *subdural*, between the dura mater and arachnoid, or it may be *subarachnoid*, between this and the pia mater. Hæmorrhage outside the dura mater is more common than within it.

Causes.—Meningeal hæmorrhage may occur at all ages. It is met with in newly-born children, but in them is due to rupture of vessels during birth, and the blood often merely descends into the spinal membranes from those of the brain. Spontaneous hæmorrhage is unknown in early childhood, but occurs at all other ages, and is more common in men than in women. Of the immediate causes, injury is the most frequent—fractures of the spinal column, blows or falls on the back which do not fracture the vertebræ, and even falls on the feet or buttocks. It is occasionally found after death from severe convulsions, epilepsy, puerperal eclampsia (in which an altered state of the blood may assist), chorea, strychnine poisoning, and tetanus. In some instances no symptoms were observed during life which could be referred to the hæmorrhage, and it is probable that the extravasation occurred only during the last moments of life. Hence it seems likely that muscular spasm, resulting from a meningeal hæmorrhage, may sometimes have been looked on as primary and independent—the cause of the hæmorrhage when it was really the effect of it. This seems certain in many cases of tetanoid spasm in newly-born children—in most cases, indeed, in which such spasm occurs almost immediately after birth and is associated with meningeal hæmorrhage. Such cases should be separated from those to which the term *tetanus neonatorum* is applied. They are further considered in the account of hæmorrhage into the cerebral membranes in Vol. II. Severe and prolonged muscular exertion has been, in a few cases, the apparent cause of spontaneous hæmorrhage. It also occurs in some diseases in which there is a hæmorrhagic tendency, such as purpuric states, and the hæmorrhagic forms of some acute specific diseases, smallpox, yellow fever, &c., and very rarely in typhoid fever apart from any hæmorrhage elsewhere. In most of these cases its occurrence has not been suspected until the post-mortem examination revealed it, and it probably occurs generally during the last moments of life. A rare cause is the bursting of an aortic aneurism into the spinal canal, after erosion of the bodies of the vertebræ, the blood being effused outside the dura mater. Hæmorrhage within the dura mater has also resulted from the rupture of an aneurism of a vertebral artery. Blood may descend into the spinal membranes from the cranium, in cases in which a cerebral extravasation, especially about the pons, escapes into the meninges; this may occur in adults as well as in the meningeal hæmorrhage that is produced during birth. Lastly, in cases of intense meningeal inflammation, ecchymoses, and sometimes considerable extravasations, have been found on both sides of the dura mater, or in the pia mater.

PATHOLOGICAL ANATOMY.—*Extra-dural* hæmorrhage comes from the plexus of large veins which lie between the dura mater and the bone. It is usually not of large extent, and the blood collects chiefly on the posterior aspect, where, in the recumbent posture, gravitation favours accumulation, and the space between the membrane and the bone is greater than in front. But sometimes the extravasation is very extensive, covering a large part of the dura mater, and in such cases it may extend through the intervertebral foramina, along the nerves. Hæmorrhage is more common in the cervical region than elsewhere, but may occur in any part. The blood is usually coagulated, wholly or partially. The dura mater is blood-stained, sometimes even through to the inner surface. The spinal cord may be compressed, but the amount of blood is not often sufficient for this.

It is easy to fall into grave error regarding the presence of meningeal hæmorrhage; when a body has been lying on the back, the veins outside the dura mater become distended with blood, and this escapes when their walls are divided in opening the spinal canal. Care must be taken to avoid the error, on the one hand, of regarding the blood thus escaping as an ante-mortem extravasation, and, on the other, of overlooking a hæmorrhage which actually exists. In all cases of acute spinal disease, in which a post-mortem examination is to be made, the body should at first lie face downwards.

Intra-meningeal hæmorrhage.—An extravasation into the sac of the dura mater (subdural hæmorrhage) may be small in quantity, or may fill the whole cavity. When small it may pass from one part to another. In subarachnoid hæmorrhage, the blood comes usually from the vessels of the pia mater; it may surround the cord for an inch or two, or may fill the whole subarachnoid cavity. Such extensive effusions are rare, except when the blood descends into the arachnoid from the cerebral membranes. Blood effused into the spinal arachnoid has been known to ascend as high as the pons, and even to break through the valve of Vieussens and get into the cerebral ventricles.* Simultaneous cerebral and spinal hæmorrhages have been known to occur from a common and powerful cause, as, for instance, in a case of puerperal eclampsia (Charrier).

In meningeal hæmorrhage, cerebral as well as spinal, the spinal fluid is often blood-stained, and thus spinal hæmorrhage may be thought to be more considerable than it really is. The spinal cord is often discoloured and compressed, and is especially damaged when the hæmorrhage is beneath the arachnoid. In extensive extra-meningeal hæmorrhage, the subarachnoid fluid may be driven away from the compressed part, and may distend the arachnoid beyond the area of compression, the limit of which is thus marked. In cases which have lasted more than a few days there are usually signs of meningitis, set up by the irritation of the blood.

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* As in an apparently conclusive case reported by Leprestre, 'Arch. gén. de Méd.,' xxii, p. 331.

SYMPTOMS.—As already stated, slight meningeal hæmorrhage is sometimes found post mortem in death from convulsive diseases when no symptoms of it were observed during life, and in these cases it is probable that the extravasation occurs during the lethal convulsions. In the cases in which hæmorrhage causes symptoms, these are, for the most part, those of meningeal irritation, and thus bear some resemblance to the symptoms of meningitis. They differ in the suddenness and violence of their onset, to which exceptions are extremely rare. These symptoms are nearly the same whether the hæmorrhage is outside or inside the dura mater.

The first indication of the lesion is usually sudden and violent pain in the back, corresponding in position to the seat of the hæmorrhage, but generally felt along a considerable extent of the back, and often severe in the loins. This pain in the back is usually accompanied by pain along the course of the nerves passing through the membrane near the extravasation, darting or burning pains, often of great intensity; they are paroxysmal in character, and between the pains there may be various abnormal sensations, tingling, &c., referred to the same parts, and accompanied by hyperæsthesia. Muscular spasm usually coincides with the pain, and involves partly the vertebral muscles, causing rigidity of the spine or actual opisthotonos, partly the muscles supplied by the nerves in which the pain is felt, partly the muscles supplied from the cord below the seat of the hæmorrhage. The convulsive movements are sometimes general. Intense pain in the back and general convulsion have been known to be the only symptoms. Occasionally there is persistent contraction of muscles, and there is usually spasmodic retention of urine. These symptoms of irritation are no doubt due in part to the irritation of the membranes (causing the vertebral pain and reflex spasm), and partly to the irritation of the nerve-roots, motor and sensory. Paralytic symptoms quickly follow—weakness and lessened sensitiveness in the limbs below the lesion. In some cases there is, at last, complete loss of motion and sensation in the lower limbs, but such absolute paralysis is not common. Loss of power at the onset of the symptoms usually indicates either simultaneous hæmorrhage into the cord or the effusion of a very large amount of blood. It occurs, for instance, when an aneurism bursts into the spinal canal.

The symptoms necessarily differ in situation according to the seat of the disease. When hæmorrhage is in the cervical region (a frequent seat), the pains are felt in the neck and arms, and the rigidity may cause absolute immobility of the neck; while dysphagia, interference with respiration, and dilatation of the pupils may be added to the other paralytic symptoms. When it is in the dorsal region, intense pain encircles the chest or abdomen. If in the lumbar region, the pain is felt in the legs, and there are early paraplegic symptoms, with loss of reflex action in the legs, and the paralytic incontinence of urine and fæces described on p. 246.

As a rule, in spinal hæmorrhage, the cerebral functions are unaffected; the unfortunate patient is conscious of all his sufferings from first to last. Occasionally, however, consciousness is lost for a short time, apparently from shock, and delirium or coma may come on, either as an indirect effect of the spinal lesion on the brain, or in consequence of a sudden increase in the intra-cranial pressure, due to the displacement upwards of the cerebro-spinal fluid. But in most cases in which cerebral symptoms coexist with those of spinal hæmorrhage, the former have been due to simultaneous intra-cranial disease; this is the case when the blood found in the spinal canal comes from within the skull, as in most cases of meningeal hæmorrhage in newly-born children (see Vol. II). In very rare cases the symptoms of spinal hæmorrhage have come on insidiously, without pain, as in a case in which extensive hæmorrhage outside the dura mater in the cervical and upper dorsal region, in a girl of fourteen, caused only very gradual weakness in the arms, and difficulty of breathing, from which she died at the end of a fortnight.* Such a case illustrates the almost universal law that there is hardly any "characteristic" symptom of a disease that is not sometimes absent, and that the absence of such symptoms has incomparably less significance than their presence.

The symptoms of meningeal hæmorrhage, in acute cases, usually reach their height in two or three hours, but sometimes not for a few days. Death may occur when the effects are fully developed; or, after the symptoms have reached their height, they may decline, to be renewed, a day or two later, by secondary meningitis. This is accompanied by some pyrexia, and may cause death; but if moderate, the symptoms of inflammation last a week or ten days, and then, in favorable cases, permanent improvement commences.

The period of the disease at which death is most common is a few hours after the onset of the symptoms. It may be due to exhaustion, in consequence of the violence of the pain and spasm, but is more often caused by the interference with respiration that results from the conjoined effect of the palsy and spasm.

Diagnosis.—In cases in which definite symptoms are produced, the diagnosis rests on the combination of sudden local pain in the back, with the other evidence of irritation of the membranes, nerve-roots, and cord, above enumerated. When the hæmorrhage is of traumatic origin, the fact that the symptoms rapidly followed an injury facilitates the diagnosis. In the extremely rare cases in which the symptoms come on insidiously, without pain, the diagnosis of the exact nature of the disease is scarcely possible.

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* R. Jackson, 'Lancet,' 1869, p. 5.

In hæmorrhage into the substance of the cord, vertebral pain is much more frequently absent than in meningeal hæmorrhage, and when it occurs is less intense and does not spread. The symptoms of injury to the cord itself are prominent from the commencement; there is sudden paralysis, it may be at first partial, and rapidly increasing; even if there is some spinal pain at the onset, the loss of power is soon recognised, not being concealed either by the severity of the pain or by spasm. In meningeal hæmorrhage, severe pain and symptoms of irritation usually precede considerable paralysis. In cases which recover, the paralytic symptoms are more persistent when the hæmorrhage is into the substance of the cord. But hæmorrhage into the cord often breaks through into the membranes, and the symptoms of both lesions are then combined.

Meningitis is distinguished by the more gradual onset of the symptoms and by the presence of fever from the first; the fact that inflammation results from hæmorrhage must be kept in mind. In myelitis, pain is commonly absent, and the symptoms of irritation are insignificant. The diagnosis from tetanus depends on the absence of trismus, on the presence of severe spinal pains, and on the sudden onset. In newly-born children the diagnosis is supposed to be difficult, but during the first few hours or days the tetanoid symptoms which sometimes occur are the result of meningeal hæmorrhage and injury to the motor cortex.

One case of spinal hæmorrhage has been recorded in which the symptoms very closely simulated those of strychnine poisoning. Violent paroxysms of muscular spasm, with intense general pain, but without spinal pain, followed by death in two hours, were apparently due to an extensive hæmorrhage into the sac of the dura mater.* Analysis revealed no strychnia in the stomach. In such a case the diagnosis would have to be determined as much by circumstantial evidence as by the symptoms, and even the post-mortem discovery of hæmorrhage could only be received as evidence of the cause of the convulsion if no other were found.

Prognosis.—In all severe cases, death in a few hours is to be feared. The prognosis only becomes less grave when the symptoms have distinctly reached their height, although there is still some danger until the period of secondary inflammation is over. Paralytic symptoms often persist for some time, but even these, in the end, may pass away. At the onset, the prognosis must be governed by the rapidity with which the symptoms develop and by the seat of the disease. Hæmorrhage in the cervical region is especially serious, because the mechanism for breathing is directly involved.

Treatment.—The first point is to secure absolute rest. Even passive movement should be avoided. Posture is also of most urgent importance. It should be on the face or side, and not on the back, so that the spine may not be the lowest part. The prone position for a few hours adds greatly to the chances of less damage. Venesection has been employed in strong individuals, with the object of arresting hæmorrhage by rapidly lowering the blood-pressure. Leeches or wet cupping to the spine, or leeches to the anus, have also been recommended in cases in which venesection is unadvisable. It is probable that the local abstraction of blood does more in this than in most internal hæmorrhages, on account of the communication between the venous plexuses and the veins in the retro-vertebral tissues; and it is probable that the wisest treatment at the onset would be scarification beside the spine, opposite the seat of pain, the blood being allowed to flow freely. This may be followed by the application of ice and by the administration of ergotine by the skin or mouth. Afterwards the bowels should be freely opened. Sedatives are usually required to relieve the pain. The stage of meningitis must be treated on the principles laid down for the management of the acute form of that disease. The residual palsy requires treatment by electricity, &c., in the same manner as the consequences of meningitis.

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* Dixon, 'Lancet,' 1879, p. 333.

In extra-meningeal hæmorrhage it seems justifiable to remove blood that is compressing the cord, by opening the canal and perhaps even washing out the canal with an astringent and antiseptic solution. Even when the blood is within the dura mater, the operation seems to deserve consideration if life is threatened.

DISEASES OF THE SPINAL CORD.

ANÆMIA AND HYPERÆMIA OF THE SPINAL CORD.

The condition of the vessels of the spinal cord after death, their fulness or emptiness, affords no indication whatever of their state during life. Inferences as to the ante-mortem state, drawn from the post-mortem condition, are altogether erroneous, with the rare exception of local change, in which the vascular condition of one part differs from the rest. Practically, such local variation occurs only as part of a local lesion, as, for instance, in the hyperæmia that attends inflammation, and the anæmia that results from pressure. Hence the occurrence of variations in the state of the vessels of the cord, and the effects that such variations may produce, are matters of inference from symptoms observed during life—symptoms that are, in themselves, open to various interpretations. Where the ground is barren of facts, theory is always luxuriant. Anæmia or congestion of the cord affords a ready explanation of symptoms the cause of which is unknown, and it is scarcely surprising, therefore, that such an explanation has been often given. But the extent to which the conditions have been invoked transcends any measure of justification, and surprise cannot but be felt at the absolute confidence and precision of detail with which these states have been assumed to exist, when there has been no tittle of definite evidence. Positive assertions always receive some credence, however unwarranted the assertions may be, and positions incapable of proof are sometimes also incapable of disproof. It would be a futile and useless task to attempt to refute in detail the various statements that have been made regarding the influence of anæmia and hyperæmia of the spinal cord. It will be sufficient to point out briefly what may be reasonably surmised regarding these morbid states.

ANÆMIA OF THE CORD.

Permanent diminution in the blood-supply to considerable areas of the spinal cord must be a result of the general narrowing of the arteries that has been met with in some cases of chronic meningitis; but it is then associated with structural changes in the organ, the effects of which obscure any symptoms that can be ascribed to the anæmia. Nothing is practically known of the special consequences of this condition. The same statement may be made of the arrest of the blood-supply to limited areas, although it may safely be asserted that loss of function of the part must be a necessary consequence. This has been also demonstrated by experiment. Suddenly produced anæmia at once abolishes function and quickly causes necrosis of the nerve-elements—some of which, however, break up less rapidly than others, and retain for a longer time the power of resuming function if the blood-supply is restored. It is possible that such local arrest of the circulation is the initial process in some organic lesions of the cord, but the fact has still to be demonstrated The question of the occurrence of embolism is referred to in a separate section.

Transient diminution in the blood-supply may conceivably be the result of imperfect embolism, but has not been proved to have this origin. It is sometimes supposed to be due to arterial spasm, dependent on the vaso-motor nerves. This has been assumed as an explanation of certain passing symptoms occasionally complained of, loss of power in the legs, and sensory disturbance, tingling, "pins and needles," &c., in the legs and arms. It may possibly also be the cause of the tetanoid cramp and tingling in the hands with which patients sometimes wake up during the night. The theory is tenable, but it is also conceivable that such symptoms are due to transient functional states of the nerve-cells of the cord. A functional derangement of these cells must be the immediate cause of the symptoms, whether such derangement is primary or is produced by spasm of the vessels. The opinion that the "nocturnal tetany," (as it may be called,) has the latter origin is supported by the fact that the occurrence of the symptom may be prevented by a small dose of digitalis, taken at bedtime.

The curious symptom known as "intermitting lameness" (which would be better termed "paroxysmal lameness") is supposed to depend on temporary diminution of the blood-supply consequent on arterial degeneration, but it is not known whether the seat of this is the spinal cord or the brain. The character of the symptom suggests the former, but its associations the latter, and it is not known that the arteries of the cord are ever the seat of atheroma in the cases in which this condition causes the cerebral symptom commonly associated with it, "dyslexia," a peculiar intermitting difficulty in reading which will be described in Vol. II. The lameness consists in a sudden weakness and stiffness of one leg, accompanied with tingling and blunting of sensibility, and of pain on an attempt to use it. The symptoms are increased by an effort to continue walking, and pass away after a rest of ten minutes or so, usually returning, on renewed exertion, again and again. The difficulty only occurs during exertion. It is familiar to veterinary surgeons as occurring in horses. The dyslexia with which it may be associated is of grave significance, as the cerebral cause seems always to have a strongly progressive character.

In *general anæmia*, such as occurs in chlorosis and pernicious anæmia, the nutrition and function of all organs suffer, and the spinal cord shares the general state. The readiness with which fatigue of the legs is induced in such conditions may be due to the spinal cord as well as to the muscles. The legs often ache, and are sometimes the seat of various disordered sensations. It is probable that these are the result of the impaired nutrition of the nerve-elements, in consequence of which their functions are readily deranged. In some patients graver symptoms occur,—weakness of the legs, sometimes of the arms also, which may increase slowly to complete paralysis. Sensation and the sphincters may also be affected. The symptoms in these cases are now known to depend upon structural changes in the spinal cord, to which reference will be made later.* The well-established fact that optic neuritis may result from chlorosis is interesting as proof of the intensity of changes in nerve-structures which anæmia may excite.

In the *quantitative anæmia* that results from loss of blood, besides the symptoms just described, paraplegia sometimes comes on, under conditions which exclude the idea of hysteria. The source of the hæmorrhage has most frequently been the stomach, kidneys, or uterus. The paralysis is usually motor only, but Leyden has observed accompanying hyperæsthesia. It may come on a few hours or days after the loss of blood, or only at the end of one or two weeks. Most cases recover. The pathological process which causes the paralysis is not known. The loss of power is comparable to the loss of sight which occurs from the same cause, in which there may be no visible morbid change, or inflammation may be found, sometimes succeeding the loss of sight, and therefore to be regarded as a result of the derangement of the nerve-elements, or of the influence to which this is due, rather than as the cause of the amaurosis.

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* See Section on "Sclerosis of the Cord from Toxic Causes."

The treatment of the symptoms due to general anæmia is of course essentially the improvement of the blood-state. Symptoms which can be ascribed, with probability, to vaso-motor spasm may be prevented by drugs that cause a more uniform arterial tone, such as digitalis or belladonna, coupled with nux vomica or strychnia, to render the effect permanent.

Hyperæmia of the Spinal Cord.

The conditions of the return of venous blood from the cord probably shield it effectually from the mechanical congestion from which almost every other organ of the body suffers when the movement of blood through the thorax is hindered. The chief mechanical congestion to which the cord is liable is that which results from the influence of gravitation, and occurs when a person is lying on the back. The distension of the veins outside the dura mater, and also of those of the pia mater, which occurs when the heart has ceased to act, and the blood is free to obey the only mechanical force which then acts upon it, sufficiently proves the power of gravitation to congest the cord. But it is not probable that gravitation has anything like the same influence during life, counteracted, as it then is, by many other forces. Some persons with imperfectly nourished cords (easily fatigued, liable to tingling, &c., in the legs) suffer from aching in the legs or spine when they lie on the back. Since this ceases when the posture is changed, it is probably due to the mechanical congestion of the cord or membranes. It is doubtful whether any other symptoms can be, with confidence, assigned to mechanical congestion.

Of active congestion our definite knowledge may be summed up in the statement that it occurs in the early stage of inflammation. We know nothing of it as an independent condition. Nevertheless many pages might be filled by the enumeration of the various symptoms of congestion of the spinal cord,—in which the unrestricted play of "scientific" fancy has elaborated a symptomatology for the separate congestion of every part of the spinal cord. Probably the only case in which we are warranted in suspecting a primary active congestion of the cord or membranes, is that in which symptoms identical with those of commencing myelitis, and following a cause of myelitis, pass away in the course of a few days. These cases will be mentioned in the account of inflammation of the cord.

An active congestion, which may be called secondary, seems to result from prolonged and violent action of the nerve-elements of the spinal cord. Thus local dilatation and distension of vessels, migration of leucocytes into the sheath and into the adjacent tissue, and even small extravasations, are found in some cases of hydrophobia, of tetanus, and of strychnine poisoning, and it is probable that a similar congestion attends all violent physiological activity, violent and continued muscular exertion, coitus, &c. Coitus several times repeated has been known to cause hæmorrhage within the cord which must be preceded by an intense congestion. Vascular dilatation, with an increased blood-supply, is the physiological attendant of functional activity in all organs, and doubtless also in the cord, but it is morbid only when excessive. It is doubtful whether it causes symptoms unless it goes on, as it probably may, to actual inflammation.

INFLAMMATION OF THE SPINAL CORD; MYELITIS.

Inflammation of the spinal cord is a common lesion in the acute, subacute, and chronic forms. It usually causes a considerable diminution of consistence, and hence the term "softening of the spinal cord" has been used as a synonym for it. But the term is not accurate, because inflammation does not always cause softening, and a diminution of consistence may occur without inflammation.*

The spinal cord differs from most other structures in that its power of recovering functional capacity after damage by inflammation is comparatively small. The symptoms persist for a long time, and are not unfrequently permanent. Hence the distinction between acute and chronic inflammation is to be drawn, not from the whole course of the disease, but from the mode of onset. It is this which indicates the character of the morbid process. *Acute myelitis* is that form in which the symptoms indicating inflammation of the cord come on rapidly and reach a considerable height in a short time, in less than two weeks. When the onset occupies from two to six weeks the inflammation is regarded as *subacute*. When a still longer time elapses before the symptoms reach a considerable degree of intensity the myelitis is termed *chronic*. There is, of course, no sharp distinction between these classes. The subacute and acute forms may be conveniently described together. Other varieties are based on the distribution of the disease. The grey matter may be affected only or chiefly, *polio-myelitis* (from πολιος, grey), a form that has distinctive characters which make its separate description desirable. When the whole thickness of the cord is affected in a small vertical extent the myelitis is said to be *transverse*. This is one of the most common forms. When an extensive area of the cord is continuously inflamed the myelitis is said to be *diffuse*; when one small area is affected the inflammation is called *focal*; when there are many foci contiguous or distant the myelitis is said to be *disseminated*, and when such foci are present in the brain and pons, as well as in the cord, the condition is spoken of as *disseminated encephalo-myelitis*. Inflammation of the grey matter, around the central canal, extending into the intermediate grey substance and to a less extent into the cornua, has been termed *central myelitis*. A further important distinction is that between *parenchymatous* myelitis, an acute affection beginning in the nerve-elements, and the *interstitial* form, which begins either in all the elements or in the connecting tissue, and in which vascular disturbance takes a prominent part. The latter is the common form of myelitis, whether transverse or disseminated, but the former is of importance on account of the limited class of symptoms to which it gives rise and of its special causal relations. This distinction is already familiar in regard to multiple inflammation in the nerves.

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* Oppenheim, Pierre Marie, and Bruns have been especially identified with objections which have been raised to the nomenclature in cases of myelitis. They allege that many cases of so-called myelitis are due to softening from vascular thrombosis and to other causes not strictly inflammatory in character. They even allege that compression or pressure myelitis is a misnomer. There is, no doubt, much truth in what they allege in reference to softening, but it is not easy to understand an objection to naming the condition myelitis which occurs, at least in many cases of compression, and the views as to what constitutes true inflammation are so conflicting that little alteration has been made in this edition in the point of view from which myelitis is regarded in earlier editions of this work.

ACUTE MYELITIS.

ETIOLOGY.—*Remote causes.*—Little is known of the influence of hereditary predisposition, but myelitis is sometimes met with in members of families in which other evidence of a neuropathic tendency can be traced. Males suffer more frequently than females. It may occur at all ages, but the form which occurs in young children is usually confined to the anterior cornua; other forms of myelitis are most common between ten and forty years.

Immediate causes.—Myelitis, acute and subacute, is readily produced by injuries to the cord of all kinds,—lacerations, bruises, and punctured wounds. It also seems to follow simple concussion, but in many of these cases there have been minute foci of structural damage. The myelitis produced by concussion is often, however, far more extensive than the primary lesion, for the latter may cause few symptoms, and a few days later a complete transverse myelitis may develop. Hæmorrhage into the cord, especially of traumatic origin, may have a similar effect; it may be followed after a short interval by extensive myelitis. In cases in which an interval of more than a few days elapses, pain in the spine may show that a morbid process is going on by which the myelitis is produced. Thus, in one case of acute myelitis, two weeks after a fall, there was continuous pain during the interval, corresponding to the seat of the later inflammation. Concussion also sets up the subacute form of myelitis, and this is especially common after railway accidents (see Traumatic Lesions).

Allied to injury as a cause is violent action of the muscles attached to the spinal column, which has sometimes appeared to be the sole agent in producing an acute inflammation in the adjacent part of the spinal cord. The disease has been known to follow a single violent contraction of the dorsal muscles, as, for instance, a successful but violent effort to prevent a fall. It is possible that there is an actual injury to the cord in these cases, but more probably inflammation is set up in the fibrous tissues of the vertebral column or nerve-roots, and spreads to the cord. It is also possible that compression of the vessels may be concerned in the mechanism. In several cases the disease has resulted from attempts to lift heavy weights. Thus a man, two days after such an attempt, became rapidly paraplegic, and ultimately died; softening of the cord was found at the level of the fifth and sixth cervical vertebræ. Slow compression (*q.v.*) is constantly attended by inflammation in the compressed part; the myelitis thus set up may have an independent course, acute or subacute, and may not be proportioned to the pressure. In cancer of the vertebral bones, for instance, very slight pressure may cause an acute inflammation; there is probably sometimes an extension of inflammation to the cord from the external disease, but such extension is not necessary for the occurrence of the myelitis; the inner surface of the dura mater is often normal. Acute myelitis also results from internal meningitis, in which some degree of invasion of the cord is almost invariable.

Another exciting cause is exposure to cold, especially when the body is heated, lying on damp grass or on snow, bathing in cold water, &c. This cause has been supposed to act by suppression of the functions of the skin, since myelitis has been produced by varnishing the skin of animals,* but it is unlikely that this is the sole mechanism. Exposure of the back to cold sometimes seems especially effective. Myelitis has been produced experimentally in rabbits by freezing the skin of the back with ether spray (Feinberg) and by painting the cord itself with Fowler's solution (Leyden). It is probable that cold usually acts by the mechanism of a morbid blood-state, such as that which causes rheumatic fever, but which certainly varies greatly in its precise character and pathogenetic power. It is also certain that local exposure frequently causes inflammation in organs corresponding to the part of the surface that is especially chilled, although this influence may be chiefly to determine the incidence of the effect of the blood-state. In myelitis from cold, local exposure can seldom be traced, and the common mechanism is probably that of a blood-state. It is instructive to compare the analogous facts regarding the causation of multiple neuritis. Over-exertion is another powerful cause, and is especially effective when combined with insufficient food and exposure to cold.

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* Feinberg, 'Virchow's Archiv,' Bd. lix. In Vulpian's laboratory similar experiments failed.

The sudden suppression of the menses and of other habitual discharges has been supposed to cause the disease. In most instances of the kind there has also been exposure to cold, to which the suppression and the myelitis may both have been due, and it is impossible to say how far the former has been concerned in the process. Thus, a girl sat on wet grass during menstruation, which was immediately arrested, and myelitis followed, a day or two later. It is conceivable that the sudden arrest of the discharge may lead to a morbid state of the blood, or modify that which a chill alone is capable of producing, and that the derangement of the uterine functions may have a direct influence on the cord and dispose it to suffer. It is noteworthy that, in the case referred to, the inflammation was in the part of the cord to which the pelvic organs are related, the lumbar region.

Functional over-action of the cord may cause inflammation in the part that is thus over-exerted. It has been already mentioned that long-continued stimulation of the sensory nerves may cause myelitis at the spot related to the nerves stimulated. As we have just seen, active function everywhere involves vascular dilatation, which, if excessive, amounts to active congestion, and, when prolonged, is attended by the escape of leucocytes that renders the condition one of inflammation. However excited, such a process has a tendency to independence of course, and to proceed beyond the degree corresponding to its cause. It is especially local functional over-action that has a tendency to cause myelitis. Thus the affection seems to be sometimes produced by sexual excesses. Occasionally, however, general over-action of the cord, as in severe, long-continued exertion, has been followed by the disease. In one case it was due to lying at rest on damp ground, after an unusually long walk on a hot day.

Toxic blood-states constitute a well-marked class of causes, and their influence is rendered the more distinct by the analogous causation of multiple neuritis. They may be acute conditions due to some organised virus, perhaps allied to those that produce the acute specific diseases.* Inflammation of the cord occasionally comes on in the course of, or after, typhus, typhoid, erysipelas, malaria, and especially variola and measles, as well as acute rheumatism and severe puerperal diseases. The onset may be during the acute disease, at any period after it has existed for about a third of its duration, or it may occur during convalescence, or a month or more after the disease is over.

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* See Buzzard and Risien Russell ('Trans. Clin. Soc.,' 1897), who describe a case of meningo-myelitis in which a diplococcus was found in the membranes, and cultures on nutrient media showed the presence of an identical organism.

Whether syphilis is a cause of acute myelitis is a question on which opinions differ. Attacks of acute myelitis are not uncommon in syphilitic subjects, and it seems on the whole probable that the mechanism is by disease of the vessels, such as is found in the pia mater in some cases.[*] The myelitis that occurs in acute specific diseases is probably due to the virus of the primary disease, to a product of it, or to a secondary infection engrafted on the first. Thus Marinesco found streptococci in the spinal cord in a patient in whom paralysis came on in the course of smallpox. The fact that the inflammation is commonly disseminated, suggests that some specific organisms have a tendency to fix themselves in the spinal cord and set up inflammation. In diphtheritic paralysis, the cells of the cord may present changes that amount to a parenchymatous myelitis. The fact that chronic changes in the cord may be a late effect of syphilis (as in tabes) makes it the more easy to conceive that acute processes should sometimes occur in an earlier stage of the disease.[†]

In another class of diseases, a special influence of the virus appears to be associated with the results of functional over-action. This effect is especially seen in hydrophobia and in tetanus. In some cases of this disease, foci of a somewhat diffuse myelitis of slight degree are conspicuous on microscopical examination. Still more intense myelitic changes are sometimes met with in the chorea of the dog, although, in other instances, the cord is normal.

Chronic blood-states and constitutional conditions may also cause acute myelitis. Alcoholism is an occasional cause, and frequently produces the subacute form. Gonorrhœa has also been shown by Leyden,[‡] Gowers,[§] Barriè, and others to be a cause of myelitis.[‖] The most potent agent of this class is gout. This is known to be a frequent cause of inflammation in various structures,—of the nerves among the rest; and cases of acute and subacute myelitis are met with, especially of the disseminated and relapsing forms, for which no other cause can be discovered. In one such case the patient had but lately recovered from a severe and prolonged attack of gouty inflammation of several intra-ocular structures. Like other chronic general causes, gout probably often acts as a predisponent when the malady is excited by some other cause.

Irritation and inflammation of peripheral organs, as the uterus, bladder, kidneys, intestine, &c., have been supposed to be an occasional cause, but this influence is exceedingly doubtful, except in the rare

[*] Sudden paraplegia occurred in a case in which there was diffuse arteritis, supposed to be syphilitic (Schmaus, 'Arch. f. kl. Med.,' 1889); see also Williamson, 'Lancet,' 1894.

[†] Bourget ('Arch. de Méd. exper.,' 1893) found acute myelitis almost destroying the lumbar enlargement, produced by the injection of the streptococci of erysipelas.

[‡] 'Zeitschr. f. klin. Med.,' 1892.

[§] 'Clinical Lectures,' Gowers.

[‖] Compare with this a condition of neuritis resulting from gonorrhœa (p. 147).

cases in which inflammation of a nerve ascends to the cord. In a considerable number of cases we are able to discover no cause to which myelitis can be ascribed. Many of these are probably due to a blood-state which may have no other consequences or may also cause simultaneous inflammation elsewhere.*

PATHOLOGICAL ANATOMY.—In recent acute inflammation of the cord, the pia mater, at the affected part, is usually red and vascular, and the cord itself may be slightly swollen. Its tissue is reddened and often marbled from distended vessels, and the distinction between white and grey matter is indistinct or lost.

The diminution of consistence which occurs in acute inflammation may be so great that the affected part is diffluent, like cream, and flows out when the pia mater is divided. It does not follow that the consistence is lessened to this degree during life; post-mortem softening occurs more readily at an inflamed part. When the vascular disturbance and extravasation are considerable, the condition is termed "red softening;" so much blood may be effused into the tissue that the part looks as if there had been an actual hæmorrhage, and in some cases an extensive extravasation does actually exist; this form is termed "hæmorrhagic myelitis." If the inflammation has existed for some time, the effused blood is altered in tint, and the colour of the softened part may be rather chocolate than red. After a longer time the changes in the blood-pigment cause the colour to be lighter, "yellow softening." In many cases the breaking up of the nerve-elements (partly, as just stated, post mortem) is out of all proportion to the extravasation of blood, so that the softening is "white." It is said that red and yellow softening may ultimately become white from the removal of the blood-pigment, but the evidence of this is inadequate. The period at which these changes of colour are found varies according to the amount of extravasation at the onset. At a later period the yellow or white opaque aspect is changed to a grey translucent appearance, from the absorption of the fatty products of degeneration, and an increase in the connective tissue. If the myelitis is very limited in extent, cavities may be formed within the cord.

The microscopical appearances in myelitis vary much according to the duration and form of the inflammation. In the fresh state the most conspicuous objects are the products of degeneration of the fibres, granules and masses of myelin, and granule corpuscles, with, in older cases, the peculiar bodies termed "corpora amylacea" (Fig. 97). With these are numerous red blood-discs, leucocyte-like corpuscles, and, after the first week, distinct cells, round, oval, spindle-shaped, or angular, with nuclei which resemble leucocytes in size, and are readily mistaken for them. Fragments of axis-cylinders may also be seen, granular in aspect, and with irregular swellings (Fig. 97).

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* Thoinot and Masselin ('Rev. de Méd.,' 1894) injected pure cultures of *Staphylococcus aureus* and *Bacterium coli commune* into rabbits, producing paraplegia, the result of spinal cord changes. The grey matter was chiefly affected, and the micro-organisms could be found in thé cord as long as six months later. They regard so-called reflex paraplegia as almost certainly the result of some infective lesion similar to that which they produced artificially.

The softened and even diffluent state of the tissue prevents us from learning much, by microscopical examination, of the condition of the parts in which the inflammation is intense and diffuse, because the breaking up of the tissue and separation of the fragments renders it impossible to obtain sections of the hardened organ. It is only where the inflammation is less intense, or less uniformly diffused, that a thorough examination can be made. The products of degeneration are best seen in the fresh state or in sections mounted in glycerine (Fig. 97). The fragments and globules of myelin in part occupy the position of the nerve-fibres from which they have come, in part are aggregated into masses.

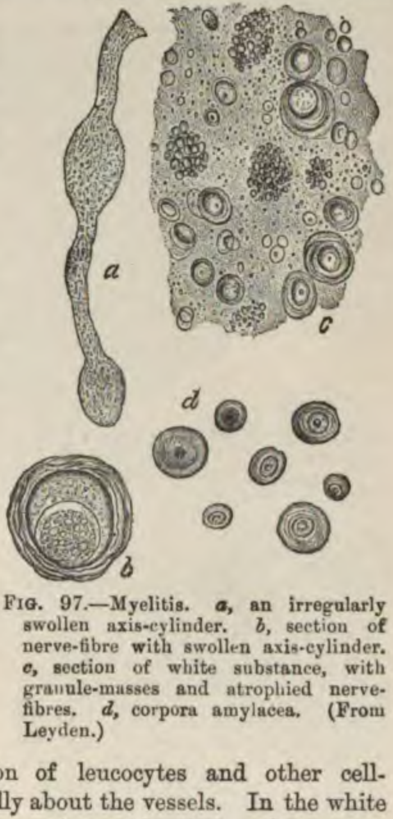

FIG. 97.—Myelitis. *a*, an irregularly swollen axis-cylinder. *b*, section of nerve-fibre with swollen axis-cylinder. *c*, section of white substance, with granule-masses and atrophied nerve-fibres. *d*, corpora amylacea. (From Leyden.)

In sections rendered transparent in the ordinary way, the fatty products of the destruction of the nerve-elements are invisible, and the most conspicuous alterations are the dilatation of vessels, which is often very great, and the accumulation of leucocytes and other cell-elements in the tissue, and especially about the vessels. In the white substance the larger vessels are conspicuous by their enlargement, and in the grey substance the dilated arteries and capillaries may occupy a considerable part of the tissue (Figs. 98, 99, A). The nuclei of the capillaries are large and numerous. The walls of the smaller arteries are encrusted with leucocytes in the early stage, and, later on, in both white and grey substance, are much thickened by cells, the nuclei of which are often elongated and more or less concentric to the lumen of the vessel (Fig. 102, D). Outside this thickened wall the perivascular sheath is enormously distended, at first by leucocytes, among which, afterwards, other cells are mingled—round, fusiform, angular, with leucocyte-like nuclei (Fig. 102). In transverse section this distended sheath may look, at first sight, like an enormously thickened wall. Red blood-corpuscles distend the vessels and are seen also in the adjacent tissue, sometimes uniformly scattered through it (Fig. 102, F), sometimes aggregated in small extravasations due to the rupture of minute vessels. The grey substance is densely set with round corpuscles, staining deeply, many of which are the nuclei of small fusiform or angular cells (Figs. 99, A, 101, B), while the intervening substance is much more granular than in health. The nerve-

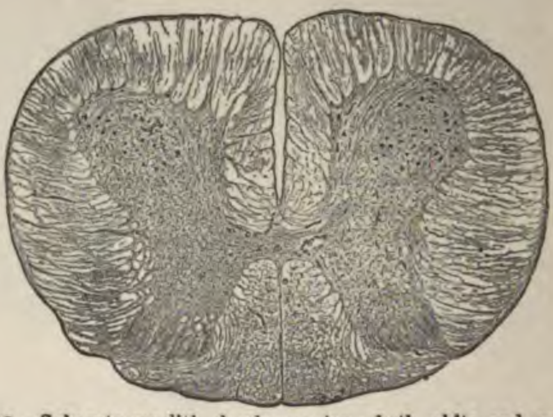

FIG. 98.—Subacute myelitis, lumbar region; both white and grey substance uniformly affected. In the latter the numerous distended vessels appear as lines; here and there a larger one is visible.

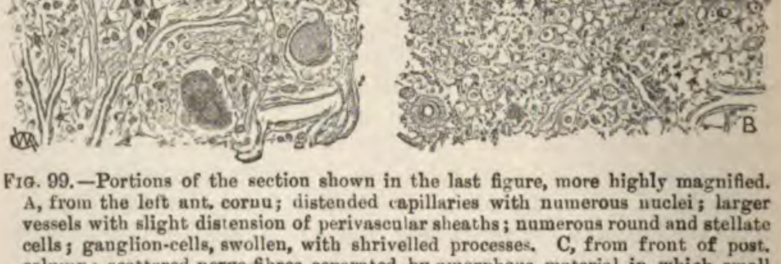

FIG. 99.—Portions of the section shown in the last figure, more highly magnified. A, from the left ant. cornu; distended capillaries with numerous nuclei; larger vessels with slight distension of perivascular sheaths; numerous round and stellate cells; ganglion-cells, swollen, with shrivelled processes. C, from front of post. column; scattered nerve-fibres separated by amorphous material in which small cells can be seen, round and irregular, some distinct, others indistinct, vessels with thickened walls. B, from near the posterior surface, shows similar changes but with more open spaces from which nerve-fibres have perished. (From sections prepared by Dr. Money.)

cells are much swollen and granular, often contain distinct globules, strongly refracting, and probably fatty. The cell-processes appear shrivelled or lost (Fig. 99, A). In slight cases the margins of the cells may be less sharply defined than in health, and the bodies then may contain large vacuoles, sometimes wholly within the cells, sometimes partly within and partly outside them in an adjacent albuminous-looking substance.* In some cases, however, especially those of disseminated interstitial myelitis of moderate degree, the nerve-cells appear little changed in hardened and cleared specimens, although a granular condition can usually be seen in the recent state.

In the white substance, cleared sections show a great increase and alteration in the interstitial tissue. The change may be uniformly distributed, or greatest in the neighbourhood of the vessels (Fig. 102, C). The increase is due in part to an amorphous material, containing nuclei here and there, some of which belong to cells such as have been described in the grey substance; and these may constitute extensive tracts between the spaces where nerve-fibres once existed (Fig. 102). In older cases the tissue may have a fibrous aspect in places. The large cells, with many processes called "spider cells," or "cells of Deiters," are often conspicuous objects (Fig. 100). The processes extend between the nerve-fibres. We have seen that their existence in the normal cord has been doubted, but in the inflamed cord, in which they are enlarged, their presence is distinct.

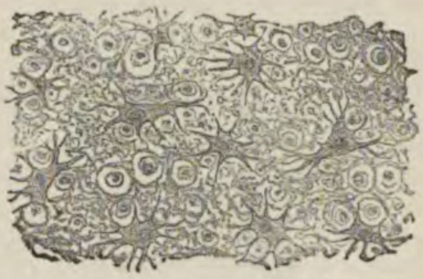

FIG. 100.—Subacute disseminated myelitis; part of the posterior column. Nerve-fibres separated by granular material and products of degeneration, and by numerous large spider cells. The myelitis in this case was attended by optic neuritis. (From a section lent by Dr. Dreschfeld.)

The changes in the nerve-fibres vary much. They always suffer destructive changes, but the products of their degeneration, above described, are scarcely to be seen in cleared specimens. The slightest change in the myelin appears to consist in an alteration in its chemical nature, in consequence of which it stains more readily than in health. Fibres are thus changed adjacent to any increase in the interstitial tissue, and they contrast with the unstained healthy fibres in the vicinity (Fig. 101, *c*). With or without this change in the myelin, there is an irregular swelling of the axis-cylinders, such as is seen on examination in the fresh state. If the fibres are divided transversely at the places where the axis-cylinder is swollen, this appears much larger than normal (Fig. 97, *b*). Examples of this will be found in most of the figures. These intense degenerative changes cause the breaking up of the myelin, and may involve the destruction of the fibre, the space occupied by it being left empty. In other cases, however, the axis-cylinder persists and remains sur-

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* The significance of this vacuolation has been much discussed. It is probable that the vacuoles form after death, perhaps during the process of hardening, but that they do not form unless the cells have been altered by disease. They have thus some pathological significance.

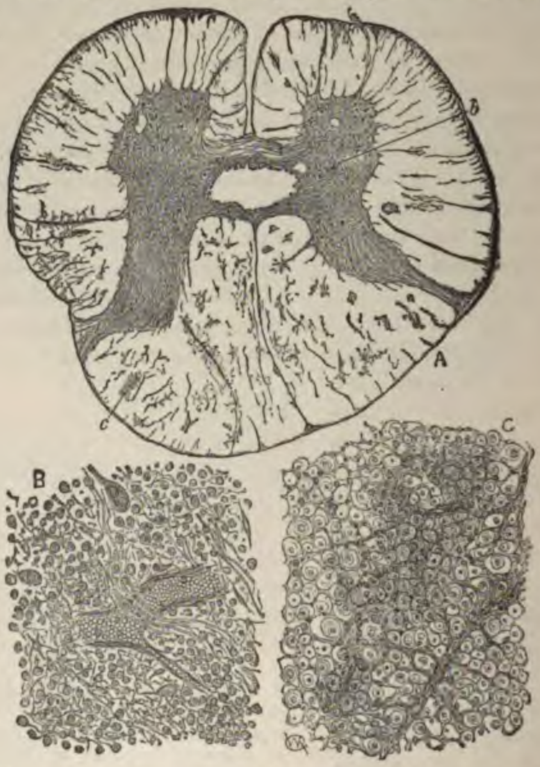

FIG. 101.—Acute transverse myelitis, fatal in three weeks. Carmine preparations. A, section through most diseased part, at first lumbar segment. Foci of inflammation scattered through the whole area of this cord, most abundant in the posterior columns (the apparent enlargement of which is probably due to the direction of the section). Dilatation of the central canal. B, from the anterior cornu at A, *b*, a distended vessel; the tissue crammed with lymphoid and other cells, among which run normal nerve-fibres. C, from the posterior column, A, *c*. The areas staining deeply are seen to consist of thickened trabeculæ, lymphoid cells, amorphous tissue, and also of nerve-fibres, the white substance of which stains with carmine while that of the neighbouring healthy fibres does not.[*]

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[*] I am indebted to Dr. Dreschfeld for the section from which these drawings are made. The patient was a girl aged twenty-three; paralysis, motor and sensory, was complete at the end of two days; the application by her mother of hot bottles to the feet caused such extensive sloughing ulcers that amputation of one leg was deemed nececsary three weeks after the onset, and the patient died the next day.

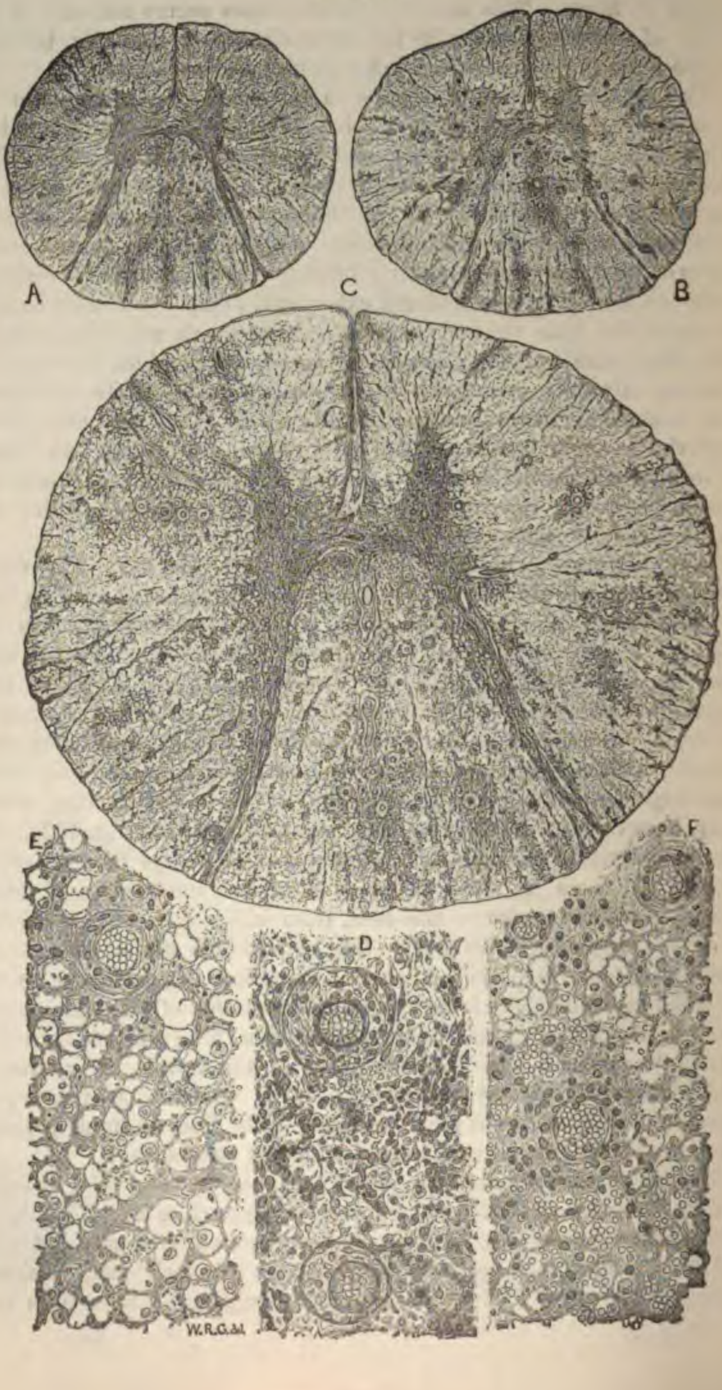
rounded by a narrow zone of myelin. Often a large number of such shrunken fibres may be found in a part which at first sight appeared devoid of fibres. This shrinking of the fibres occurs not only as the result of pressure on them by the connective-tissue elements, but also where there is little interstitial change and no compression.

The relative amount of change in the nerve-elements and in the interstitial tissue varies very much. In some cases the interstitial changes are evidently primary, and the fibres at first present little change in slight cases, but afterwards are altered secondarily, being narrowed, and even destroyed, as they are surrounded and enclosed by the products of the inflammation. In other places, or even other parts of the same section, there is but little increase in the neuroglia, although the fibres have suffered extensively; empty spaces are limited by septa but little thicker than normal, although with more nuclei, and often with numerous granules, the nature of which is uncertain. Occasionally there is a more diffuse change in the white substance; the neuroglial tissue is increased by small cells and amorphous material in such a manner that all appearance of septa and spaces is lost; a few nerve-fibres are included in it, and the spider cells are very conspicuous objects, their processes apparently representing the septa that exist in the normal condition (Fig. 100).

The distribution of these changes is very variable. The whole thickness of the cord is uniformly affected in pressure-myelitis (see "Compression of the Spinal Cord"). In transverse myelitis the changes may be so intense in all the structures at the affected level that the cord is diffluent, and, as already stated, only separated products of degeneration and inflammation, and blood-corpuscles are to be seen. In slighter cases the alteration is seldom distributed uniformly, and in either a single section, or a series of sections taken near together, each part may present variations in the damage, which may, however, be such that no part escapes in all (see Fig. 102). In meningo-myelitis the periphery of the cord is always most affected, and in old cases the thickened pia mater may send tracts of dense tissue into the interior of the cord, from which branching processes of tissue may pass on all sides. The condition is similar to that met with in chronic myelitis. A similar extension is often conspicuous in cases in which there is no special inflammation of the membranes, and the interstitial changes often extend from the septa that normally pass into the cord and from the walls of the vessels, the course of which may thus be marked out when the vessels cannot be seen. Hence the white columns may be broken up by lines of tissue passing from the surface of the cord towards the grey substance.

In disseminated myelitis the foci of inflammation may exist through a wide extent of the cord, and appear on the surface as reddish-grey areas, closely resembling those of disseminated sclerosis, but less sharply outlined. Under the microscope, in the early stage, smaller foci stud the grey and white substance irregularly, sometimes ex-

FIG. 102.



tending into larger tracts. They may be most abundant in the grey or the white substance. At first the vascular changes are the more conspicuous, but afterwards the evidence of destruction of nerve-elements and the inflammatory products, exudative and cellular, described above, characterise these foci, while ultimately a condition of irregular focal sclerosis remains. In central myelitis there is a great increase in the nuclei about the central canal, with vascular changes in the commissure adjacent, but there may also be evidence of slight widely diffused myelitis. It is necessary to remember that the nuclei around the central tissue vary much in number, and that it is easy to mistake a normal for a diseased condition.

Little is known of the morbid appearances in the parenchymatous forms, except that in a variety of anterior polio-myelitis, and in diphtheritic and other toxic paralyses, the nerve-cells present granular changes, and the interstitial grey substance may be but slightly affected.

The nerve-roots coming from a much inflamed part are usually damaged, and may present histological changes similar to those within the cord—distension of vessels, increase of nuclei and of connective tissue about the vessels, breaking up of the myelin, swelling of the axis-cylinders. Degenerative and neuritic changes may descend the motor nerves, although rarely to the same degree as in polio-myelitis, except in the cases in which there is extensive inflammation in the lumbar or cervical enlargement.

From the foci of inflammation, ascending and descending degenerations pass along the tracts of long fibres that have been already described, downwards in the pyramidal tracts, upwards in the posterior median columns, the direct cerebellar tracts, and the antero-lateral ascending tract. In cases of transverse myelitis, moreover, changes that are definitely inflammatory in nature may be traced in certain tracts for a short distance above or below the chief lesion; they may accompany the secondary degeneration, or may be found extending along a tract that degenerates in the opposite direction. Thus distinct inflammation may pass, for a short distance, up the

FIG. 102.—Acute transverse myelitis; from a case fatal in a month. A, B, C, neighbouring sections from the inflamed part, in the mid-dorsal region, to show the varying distribution of the inflammation. (Stained with aniline blue-black.) Distended vessels are all surrounded by thick zones of clear tissue, in places divided obliquely, while outside this, ramifying tracts of darker tissue extend into the white substance. In D, from the front of the posterior column, the changes are shown more highly magnified. The zone around the vessels is seen to consist of the perivascular sheath distended by cellular elements, round, oval, fusiform, angular. The white substance is crammed with similar cells, and hardly any trace of nerve-fibres or of normal structure can be perceived. In E, from a less affected part of the lateral column, stained with carmine, the alveoli remain, although the tissue between them is thickened, and in many places studded with refracting granules and larger nuclei. In many spaces nerve-fibres are seen much smaller than normal in consequence of the wasting of the white substance. F represents a similar condition, with fewer nerve-fibres, and extensive infiltration of the alveoli with blood-corpuscles. (From sections prepared by Dr. Money. For the specimen I am indebted to the late Dr. Hadden.)

pyramidal tracts, so that, an inch or two above the upper limit of the general inflammatory changes, an ascending inflammation of these tracts may be combined with an ascending degeneration of the tracts that always present secondary ascending degeneration.

If the patient survives, the new interstitial tissue slowly undergoes changes which give it a fibroid aspect, although for some time cell-forms may preponderate in it. We know very little of the nature of the process that occurs in the cases which slowly recover, and subserves restoration of function. The fact that a long period of total palsy may be succeeded by the slow return of considerable power, shows that even greatly damaged fibres may regain functional capacity. It is easy to conceive that the fibres which are only so far changed that the axis-cylinders are swollen, and the myelin stains readily, may speedily recover. We can also understand that the fibres in which a very narrow layer of myelin remains around the axis-cylinder may also regain the power of conducting. We must presume that in these fibres there is no interruption of the axis-cylinders, but there are cases in which we can scarcely believe that the axis-cylinders retain their continuity, although conducting capacity is ultimately restored. An absolute motor palsy of twelve months' duration must depend on changes that involve an absolute interruption of the fibres, and yet in such a case some return of power may occur. Is this effected by a growth of new fibres such as occurs in nerves? In the lower animals such a growth of fibres has been proved to occur; in man it has not yet been demonstrated, but it is difficult to conceive any other explanation of the clinical facts. The very interesting appearance presented by the section of a cord shown in Figs. 103, B, and 104 suggests strongly a process of regeneration. The cord was crushed in the mid-dorsal region by a fracture of the spine, and motor and sensory paralysis remained absolute, up to the level of the lesion, till the patient's death, six months later. At the seventh cervical (Fig. 103, A) the cord presents only the usual ascending degenerations, but at the eighth cervical (Fig. 103, B) opaque tracts extend through the grey and white matter, and adjacent to these there is everywhere an extensive destruction of nerve-fibres. In these tracts within the lateral columns a large number of very minute nerve-fibres can be seen, each consisting of a fine axis-cylinder surrounded by myelin (Fig. 104, C). The fibres are smaller than any met with in the normal cord. The appearance is as if there had been an irregular ascending myelitis, which had extended up the cord as high as between the seventh and eighth cervical segments, and from the lower extremity of the normal fibres there had occurred a growth of new fibres such as effects the regeneration of nerves.* We seem to have here an actual process of renewal of fibres that had been destroyed by such inflammation as has caused the empty spaces

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* For these sections I am indebted to Dr. F. G. Penrose. Unfortunately I have been unable to obtain any other sections of this part of the cord.

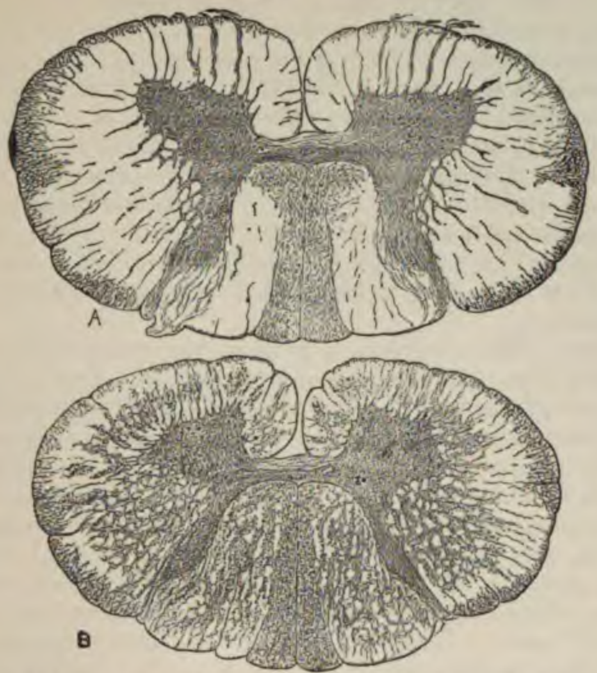

FIG. 103.—Ascending myelitis from fracture of spine completely destroying the cord in the mid-dorsal region. A, from the seventh cervical, presents only ascending degenerations in post. med. col., direct cerebel. areas, and ant.-lat. ascending tract. B, from the eighth cervical, presents also numerous areas of finely granular aspect under a low magnifying power, the finer structure of which is shown in Fig. 104.

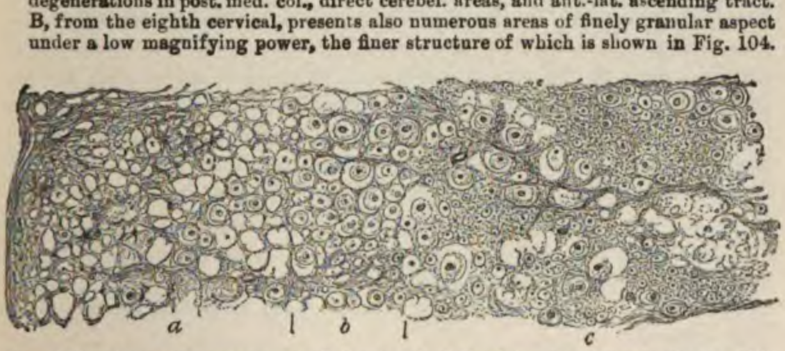

FIG. 104.—From the outer part of the front of the lateral column in B, Fig. 103. *a* is the antero-lateral ascending tract, the fibres completely degenerated, except a few, probably of other nature and belonging to *b*, a narrow zone, nearly healthy, of the antero-lateral ground fibres. *c* represents the outer part of one of the finely granular tracts. Under the higher magnifying power these tracts are seen to consist of very minute nerve-fibres with a granular material between them in which few cell-forms can be distinguished. Adjacent to these tracts there has apparently been extensive destruction of the fibres, large spaces being occupied by products of degeneration and appearing empty in the cleared section.

in the vicinity. If this is a correct interpretation of the appearances they constitute proof that, even in the human cord, it is possible to have a new formation of destroyed fibres. That pyramidal fibres may

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occupies only a few minutes. The legs are found suddenly to be heavy and tingling, the sufferer sits down on a chair for a quarter of an hour, and then finds that he cannot stand. Such rapid onset resembles that of spinal hæmorrhage, and it is probable that in most of these cases there is hæmorrhage in addition to inflammation—"hæmorrhagic myelitis." Such cases are, however, sufficiently rare not to interfere with the diagnostic rule that a sudden onset means a vascular lesion,* and that the characteristic onset of myelitis is rapid but not sudden. Now and then the onset is in the night, during sleep; a patient goes to bed well, and wakes up in the morning with complete paraplegia. On the other hand, the onset of the paralysis may occupy several days, or even a week; when longer than this, from ten to forty days, the myelitis is to be regarded as subacute. Occasionally the onset is by a series of sudden attacks of weakness at intervals of a few hours or days, or even weeks. In the latter case there may be some recovery from one attack before the next comes on. Frequently there is gradual partial loss of power for a few days, and then complete paralysis comes on rapidly; the first symptoms may be slight, and their nature only recognised when the disease has developed.

When the paralysis has reached its height, and has ceased to increase, it is usually complete; sometimes it is incomplete, the limbs can be moved, but with little power, or the patient may be able to contract certain muscles, but not to move the parts to which they are attached, perhaps only to move the toes. The distribution of the paralysis depends on the position and extent of the disease. In the majority of cases, the legs and lower part of the trunk only are affected, because the dorsal region of the cord is the most frequent seat of myelitis. If it is incomplete in the legs, the flexor muscles usually suffer more than the extensors, although this may not be apparent as the patient lies in bed, because the leverage at the knee renders necessary considerable power to extend the joint against gravitation. When the disease is in the cervical region the arms as well as the legs are involved, and the arms are sometimes paralysed before the legs. If the lesion is extensive in the cervical region, the damage to the grey matter and root-fibres causes atrophic palsy in the arms, while the legs are the seat of simple and ultimately spastic paralysis. In such cases the intercostals are paralysed, and breathing is carried on by the diaphragm. Myelitis above this level, if complete, abolishes all respiratory power, and necessarily causes death with great rapidity; but occasionally, in partial inflammation, the diaphragm may escape, or the intercostals retain some power and life be preserved. In such rare cases the arms as well as the legs may be the seat of simple palsy, and the neck muscles may become atrophied. This was seen in one patient, in whom the sterno-mastoids and the upper part of each trapezius were wasted, while the arms as well as the legs presented an increase of myotatic irritability. In such cases disseminated inflammation may spread into the medulla, the pharynx and the tongue may be affected, vomiting may occur, and the heart's action be interfered with. The range of motor symptoms, when the disease is at different heights, is indicated in the table of functions (p. 252).

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* Indeed, it is possible that these cases constitute no exception to the rule, the myelitis being set up by vascular obstruction.

Sensation is frequently impaired as well as motion. At the onset, indeed, it is involved in some degree in all cases of general transverse myelitis. In severe cases it is absolutely lost up to the level of the lesion. The upper limit of loss is often oblique, being higher in front than behind. In slighter cases the loss may be partial, and only certain forms of sensation may be impaired, either in consequence of the position of the disease, when touch or pain may be lost alone, or of its slight degree, when touch is generally impaired and painful sensibility preserved. Occasionally there is general hyperæsthesia in the limbs at the onset. At the level of the lesion there is usually a zone of hyperæsthesia, corresponding to the distribution of the nerves that pass through the upper part of the affected region, i.e. through the lowest, merely irritated, portion of the cord above the disease. The hyperæsthesia may readily be detected by passing a hot sponge down the spine; the sense of warmth changes, at the hyperæsthetic zone, into one of pain. Corresponding to this zone of hyperæsthesia there is often a sense of painful constriction, a "girdle pain," which may persist for a long time, even after the loss of sensibility below the lesion has passed away. Occasionally it only comes on some time after the onset, probably from cicatricial compression and irritation of the root-fibres. Its position varies with that of the disease. It is most commonly felt between the umbilicus and ensiform cartilage, sometimes around the lower part of the abdomen, about the anus, or in the legs. It is of much practical importance because it is evidence of the existence of organic disease, and also of the upper limit of the lesion. It is sometimes a very early symptom. Any initial pain in the limbs usually ceases when sensation becomes abolished. When pain persists in the legs, it is often less when these are flexed.

The state of reflex action varies, and depends on the position of the disease, in accordance with the laws already stated (p. 262). An acute lesion in any part of the cord may cause an initial inhibitory loss of reflex action in the parts below; but if the lesion is above the lumbar enlargement, reflex action returns in the course of a few hours. Frequently there is no initial depression. Subsequently the reflex action becomes excessive, that from the skin rapidly, that from the muscles more slowly. Ultimately each attains a high degree of exaltation. If the disease involves the lumbar enlargement all forms of reflex action are lost.* When in the cervical region it may be lost in the arms, excessive in the legs. In dorsal myelitis the trunk-reflexes are often impaired, and may give important information as to the seat of the disease. The abdominal reflexes should be examined at various levels, and the greater impairment of these on one side than on the other may reveal clearly the unequal distribution of the lesion and its vertical extent.

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* See, however, the facts stated on p. 264.

The muscles of the legs are sometimes at first flabby and toneless during the stage of initial depression of reflex action, doubtless from the same influence. This condition soon passes off if the lesion is above the lumbar enlargement, and as reflex action returns the muscles regain their tone. If, however, the myelitis involves the lumbar enlargement in considerable degree, the muscles of the legs remain flaccid, and lose all influence on the posture of the limbs. The feet fall into an extended position, so that the instep is in a line with the tibia (Fig. 105). The muscles also waste rapidly, and often present the reaction of degeneration. The wasting in these cases is often extreme. In many cases of myelitis above the lumbar enlargement there is a slighter and slower wasting of the muscles, without loss of reflex action, and generally with excess of the muscle-reflexes; the ultimate amount of wasting may be considerable, but it is never so great as when the lumbar grey matter is diseased. There is never the reaction of degeneration, but only a slight change in irritability, similar to each current; first an increase, which after some weeks or months gives place to a slight diminution.

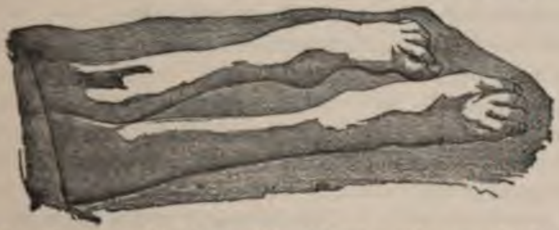

FIG. 105.—Posture of feet in myelitis of lumbar enlargement with rapid wasting of muscles.

The sphincters are usually affected from the first, except in very slight cases, and they often afford the earliest indication of the commencing lesion. There is usually first retention of urine, which not rarely exists for several hours, or even for a day or two, before other symptoms are added to it. There is afterwards incontinence, overflow or reflex (see p. 247), when the disease is above the lumbar region; if the lumbar centre is diseased there is persistent incontinence from the first without retention. In the former case there is involuntary action, in the latter inaction, of the sphincter ani, and in both cases incontinence of fæces. This may be inconspicuous in the early period, on account of constipation, due to intestinal paralysis. In cases of partial myelitis, as of the anterior cornua, the sphincters are often unaffected. The urine is frequently alkaline in reaction, and the change sometimes comes on so rapidly that it is evide the result of an alteration in the secretion. In all cases of reten decomposition in the bladder increases the alkalinity. Cystitis c follows, so that the urine contains pus.

The temperature of the paralysed limbs is usually at first raised or two degrees above that of the mouth, but it afterwards falls, remains a degree or so lower than that elsewhere. The skin is c ry, sometimes covered with sweat. Its nutrition often suffers, [illegible] result. In some cases they are due to long-continued care and neglect, but they may occur early and intensely when disease affects the lumbar enlargement or irritation extends dow from above. The least pressure on the skin may then cau bulla containing sanious liquid, and sloughing occurs with readi especially over the sacrum. Now and then there is effusion into [illegible] joints. Occasionally the tendency to sloughing of the skin i [illegible] that the lesions appear to be spontaneous.

Such trophic disturbances sometimes occur in the cellular tissu the lower part of the body, especially in the neighbourhood of bladder and rectum. A remarkable example of such disturb was presented by the case from which Figs. 98 and 99 are ta Symptoms of subacute myelitis were followed by those of celluliti the lower part of the abdomen, on account of which the patient admitted into University College Hospital under the care of my [illegible] Mr. Marcus Beck. Subcutaneous emphysema developed al [illegible] and suppuration occurred near the rectum. In co [illegible] this inflammation the patient died, the cord symp [illegible] increased; after death no lesion of the intestine or [illegible] the cellulitis could be discovered. It is probable tha [illegible] of the trophic influence of the cord facilitates the [illegible] cystitis from retention of urine, and intensifies its eff [illegible] Ulceration of the bladder, which sometimes occu [illegible] rapidity, may be thus produced, or a small absce [illegible] wall of the bladder, and thus an opening may be [illegible] peritoneal cavity, and fatal peritonitis may be indu [illegible] fistula may be developed in the same way. Ul [illegible] and consequent extravasation of urine, ha [illegible] similar conditions.* In another case the ir [illegible] bladder was intense, and suppurative cellulitis [illegible] and set up a fatal peritonitis; the kidneys [illegible] suppurative inflammation.† These effects [illegible] be prevented.

[illegible] is in the cervical region the pupil may b [illegible] myelitis, optic neuritis has been observe [illegible] complication to cause it.‡ It is probab

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[illegible] Hosp. Rep.,' vol. x.

[illegible] 'Trans. Ophth. Soc.,' 1883.

[illegible] present in the case of disseminated myelitis result of the inflammation of the spinal cord, but is an associated and similar lesion, the result of the cause of the myelitis, which, in such cases, is probably a blood-state. It is noteworthy that most of the cases thus accompanied have been instances of disseminated myelitis, a form that suggests a cause acting widely on the nervous system. In one case[*] the optic neuritis reached its height some weeks before the occurrence of the first spinal symptoms, and in the cord were two separate and distant foci of inflammation; in another the neuritis was found a fortnight before the onset of disseminated myelitis.[†]

In cases of rapid development the degree attained by the symptoms at their onset may not be exceeded. In some cases a slower increase or extension ensues in the course of a few days. The myelitis, beginning below, may ascend the cord, and the arms and respiratory muscles may gradually become involved. The upward extension may be steadily progressive, or it may occur in successive separate attacks. In the latter case, it seldom extends above the dorsal region. Or inflammation, beginning in the dorsal region, may slowly descend into the lumbar enlargement. The extension may be by the grey matter, especially of the anterior cornua, or by the posterior region. In each case the extension downwards is indicated by loss of the reflex action present in the earlier stage: in the one there is rapid muscular wasting with loss of faradic irritability; in the other the muscles retain their nutrition and irritability, but sensation, if before present, becomes lost.

The constitutional symptoms, which have been mentioned as attending the onset, vary greatly in their severity and course. They are, as a rule, less in the simple transverse myelitis than in the disseminated form. In most cases they reach their height on the second or third day after the onset of severe symptoms. Thus in one case the temperature was normal on the first day of indisposition, when the patient complained only of slight headache and slight numbness of the legs. During the next three days these gradually became completely paralysed, and the temperature was successively 102°, 103°, and 104·5°, and then gradually fell to normal at the end of a week. In another case, in which the toxæmic state was part of that of measles, the temperature rose from 100° to 103·8° on the first day of the symptoms of myelitis, and on the next day to 104·2°, but fell (partly from antipyrin) on the third day, on which death occurred, to 100·2°.[‡] In less acute cases, in which foci of inflammation develop progressively with spreading palsy, during one or two weeks, moderate irregular pyrexia, 99° to 101°, may go on as long as the disease is increasing, and may be renewed at any subsequent relapse. The temperature is also raised

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of Dr. Dreschfeld from which Fig. 100 is taken, and also in that of Sharkey and Lawford just mentioned. See 'Medical Ophthalmoscopy,' 3rd ed., p. 189.

[*] Sharkey and Lawford, op. cit.

[†] Achard and Guinon, 'Arch. de Méd. exp.,' 1889.

[‡] Barlow, 'Med.-Chir. Trans.,' 1887, p. 76.

by any complications, and especially by cystitis. On the other hand, in simple cases, with a single focus of inflammation, the initial elevation may be trifling, and after the first day or two the temperature remains normal.

If the patient survives, the course of the symptoms varies according to the intensity of the lesion. The paralysis, motor and sensory, may remain complete. More frequently sensation is recovered after a few weeks or months, while motor power continues absent for a much longer period. The excessive reflex action may lead to the gradual development of spasm in the legs. This is especially related to the increase of the muscle-reflex action which follows the secondary descending degeneration of the pyramidal fibres. Ultimately the condition is that termed "spastic paraplegia," and described in detail in another section. The occurrence of this spasm is not incompatible with the recovery of some voluntary power. The muscles in these cases are often well nourished, and may even increase in size; the spasm constitutes a powerful stimulus to their growth. Spasm may, however, co-exist with the slow moderate wasting described above (p. 265), but is rarely intense in these cases.

In some cases, flexor spasm, after a time, takes the place of the extensor spasm, at first in transient attacks but at last permanently, and becomes fixed by contracture and shortening of the flexor muscles. It may be so great that the knees are against the abdomen and the heels against the nates. This substitution of flexor for extensor spasm occurs chiefly in cases in which the paralysis is absolute, and the organic disease of the cord so considerable and of such duration as to make recovery impossible. It is therefore a very grave sign.

Death may occur early in the disease from respiratory paralysis, or from the extension of disseminated myelitis into the medulla oblongata; subsequently it may be due to various effects of the trophic disturbance, acute or chronic, and to blood-states induced by bedsores or by kidney disease resulting from cystitis, &c., mechanisms that have been already mentioned in the account of the symptoms. Improvement, when it occurs, is usually slow, but continues for a long time, and recovery may be complete in cases of moderate severity. When there is much damage to the cord, however, recovery is often imperfect, and some weakness remains, accompanied either by spasm or by wasting. It is rarely that no improvement occurs; occasionally, however, the palsy remains absolute, although life is prolonged for years. In some cases improvement occurs, slight or considerable, and is followed by a relapse, which leaves the patient worse than the first attack; and this may occur again and again, each relapse being the expression of a fresh extension of the inflammation in the cord. This relapsing course is met with especially in the disseminated form, and in the subjects of gout, either acquired or inherited. Other cases, again, present what may be termed a recurrent course, improvement, and perhaps a considerable degree of recovery, continuing for months, when a fresh attack occurs. On the other hand, in many cases, especially of simple transverse myelitis, when improvement is established it goes on, and there is no tendency to recurrence.

Varieties.—The different pathological forms of myelitis are attended by some differences in the symptoms. Those of *transverse* myelitis have been described as the most common and typical manifestations of the disease. *Focal* myelitis, in which there is a single spot of inflammation, not extending across the cord, may occur in various parts of the cord; but, on account of the slight degree of severity of the symptoms, little is known of them except when the focus of inflammation is of some size or is situated in one anterior cornu (as a variety of polio-myelitis), because such cases are rarely fatal. It is, however, probable that this variety is not uncommon. Cases are occasionally met with in which symptoms of very limited range come on acutely, and may reasonably be ascribed to such a lesion. They may be one-sided, and various in character, often being limited to a single limb.

A much more frequent focal form is *disseminated myelitis*, in which there are several foci of inflammation in the same or different parts of the cord. The onset of this form is often subacute, and constitutional symptoms are frequently absent. The most important special symptoms are those which indicate interference with the central functions of the cord in more than one locality, but the combinations produced are very variable. In many cases the several foci of inflammation develop successively, not simultaneously, and then we have a corresponding succession of symptoms. Thus a myelitis in the cervical region, causing atrophy of some groups of muscles in one arm and paralysis of the corresponding leg, may be followed by paralysis of the other leg with such loss of reflex action in it as shows a separate focus of inflammation in the lumbar enlargement, and this again by the development of a girdle pain corresponding to the middle of the dorsal region, from a fresh area of inflammation in that situation. When numerous foci of myelitis occur in the dorsal region, as is not uncommon in syphilitic cases and after injury, the symptoms may closely resemble those of a transverse myelitis, because by one or another of these foci each of the elements of the cord is interrupted. The only distinction may be the extensive impairment of the trunk-reflexes, or the detection of loss of irritability in the corresponding muscles. Closely allied to disseminated myelitis is the condition known as *disseminated encephalomyelitis*, described by Leyden over twenty years ago under the name of acute ataxy. It is characterised by the presence of small foci of change in the cord, pons, and cerebrum. In some cases such foci have only been found in the cord. The symptoms are those of interference with the functions of the parts in which the foci are formed, *e. g.* defects of speech and articulation, tremor and ataxy in the legs and arms, tremor of the head, and exaggerated reflexes associated with a spastic gait. The condition seems to be frequently dependent upon some antecedent infectious disease or other toxic blood-state, *e.g.* inhalation of a poisonous gas. It is more frequent in children, is variable in its course, sometimes followed by complete and fairly rapid recovery, in other cases running a more chronic course with alternating relapse and recovery. Sometimes it leads to permanent disablement or impairment of motor functions. The sphincters may escape, and the mental condition is not as a rule impaired, except perhaps at the onset, which may be characterised by unconsciousness and delirium. According to some observers, the condition may merge into one not distinguishable from disseminated sclerosis. Leyden, however, asserted that disseminated encephalomyelitis has no tendency to extend.

In the rare form of *diffuse central myelitis* there is usually rapid loss of power, of sensation, and of reflex action, considerable elevation of temperature, speedy trophic disturbance, and often death at the end of two or three days. Rarely sensation and reflex action have been lowered and not lost. On the other hand, the loss of sensation is said to be sometimes absolute, when motor palsy is incomplete. The symptoms have begun in arms and legs simultaneously, or in either of these, and have accordingly spread upwards or downwards. Practically nothing is yet known of any slight non-fatal forms of this variety. *Hæmorrhagic myelitis* is scarcely a special form, since any acute inflammation of the cord may be attended by a sudden extravasation of blood. Its manifestation is the sudden onset of severe symptoms after slighter disturbance, such as indicates a commencing myelitis.

Parenchymatous myelitis is known only in a few forms, and we have much yet to learn regarding it. In diphtheritic paralysis there is an acute degenerative change in the nerve-elements, especially in the nerve-cells, which must be regarded as essentially a lesion of this form. In some cases of polio-myelitis the motor nerve-cells suffer primarily, and the interstitial tissue is not affected except in the severer cases of this form. In all inflammations, whether parenchymatous or interstitial in nature, all the tissues of an organ tend to be involved if the process is acute. Other symptoms that are of importance, as suggesting this variety, are those of acute ataxy, clearly dependent on an affection of the cord occasionally met with. Thus a married woman, who was certainly the subject of syphilis, and had presented secondary symptoms a year previously, found one day that she could not walk so well as usual, and the next morning could not stand, on account of extreme inco-ordination in the legs. When she tried to use them, the resemblance to the extreme degree of locomotor ataxy was perfect. The knee-jerk was normal on the right side, almost lost on the left. The left leg was thought to be a little weak, but in a day or two its power was good. For the first few days there was great hyperæsthesia of the lower part of the trunk and legs, but at the end of a week this was reduced to a band at the level of the lower half of the abdomen, where even a touch occasioned pain; subsequently a girdle pain developed in this region. Her condition lasted unchanged for two months, and then, iodide and belladonna being given, slowly improved; but it was not until four months after the onset that she could walk a little, and then only in a highly ataxic manner. Her recovery was ultimately perfect.

In another group of cases, of which I have seen several instances, inco-ordination comes on acutely in one arm, and may be accompanied by complete muscular anæsthesia, so that the power of estimating weights is absolutely lost, a poker and feather seeming alike, although cutaneous sensibility is perfect. The condition has reached its height in the course of a few hours, remained complete for weeks, and slowly passed away, in the cases I have seen. The precise nature is uncertain, but provisionally it seems best to class them together as cases of acute myelitic ataxy.* Their dependence on an organic spinal lesion seems certain, and the onset of this is incompatible with any other than an inflammatory process. It is possible that some of these cases are due to a focus of ordinary inflammation, so situated as to affect structures of a common function, while others may be parenchymatous.

Pathology.—Very little is known of the pathology of acute myelitis,—that is, of the actual mechanism by which it is produced, of the nature of the causes to which it is due, and of the relation of the morbid process to its causes. The spinal cord differs from most organs in its great liability to primary inflammation, in which it presents a remarkable contrast to the brain. The liability is not equally distributed in the cord, but is least in the parts nearer the brain, less in the cervical than the lumbar enlargement, and less in these than in the dorsal region, so far as the most common form is concerned. The special liability of the dorsal region to transverse myelitis may be associated with the readiness with which this part undergoes post-mortem softening, but we are ignorant of the conditions by which this liability is determined. The disseminated form has less tendency to be localised in any special part. A question of considerable interest is whether acute inflammation of the cord always begins as such, or whether, in any case or form, it is set up by vascular obstruction, such as thrombosis in a minute vessel. It is conceivable that such an initial lesion may ultimately disappear in the intense inflammation it excites.† Myelitis may be readily produced by the arrest of the blood-supply even for a short time, as numerous experimenters have shown, but their researches throw little light on the disease as it occurs in man; the effect is to produce first necrotic breaking up of the nerve-elements, and long after this the vascular lesions of ordinary interstitial myelitis. The processes are essentially different. But the experiments show that the lumbar enlargement is thus damaged with special readiness, and this suggests that the proclivity to disease of this part is due to its inherent susceptibility.

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* For a typical case apparently of this character see Campbell Thomson, 'Lancet,' 1897, vol. ii, p. 1586.

† Williamson ('Lancet,' 1894) has shown that myelitis is sometimes the result of obstruction occurring in vessels with syphilitic endarteritis.

At present the questions of chief interest are the extent to which the disease is due to a morbid blood-state, the probable nature of such a cause, and the mechanism by which it acts. The chief facts that throw light on the subject have been mentioned already, the most important being the occurrence of myelitis in the acute specific diseases (in which we must ascribe it to the organised virus of the primary malady itself, to some secondary but associated organised virus, as the staphylococci found in Marinesco's case of myelitis occurring after smallpox, or to some chemical toxin, the product of organisms), and the evidence of an influence acting throughout the system, afforded by the wide extent of the disseminated form and its occasional association with disease elsewhere, such as optic neuritis. It is to this form that our knowledge chiefly relates. Of the pathology of simple transverse myelitis we have only the fact that it often follows cold, which brings it into closer analogy with the simple inflammations of other organs.

The knowledge that has been gained of the influence of toxic blood-states on the peripheral nerves gives us the help of analogy in conceiving that myelitis may often have the same origin. But a difference exists in the fact that the multiple neuritis so produced is commonly parenchymatous, while the myelitis is conspicuously interstitial. Of the pathology of parenchymatous myelitis we know almost nothing. The question of causation will be again alluded to in considering the special inflammation of the grey matter.

The chief relation of the symptoms to the lesion has been considered in the general account of the symptoms of spinal cord disease, and those relating to the destructive and regenerating processes have been mentioned in the section on pathological anatomy. We may, however, further note that the vascular changes, which take so large a share in the process of interstitial inflammation, as seen, for instance, in the disseminated form, must extend the process. The escape of leucocytes into the sheaths tends to interfere with the lumen of the vessel and the flow through it, while their accumulation in the tissue must entail local destruction of the nerve-elements. If the cause of the myelitis is a persistent influence, such as the poison of a progressing specific disease, or the agent, whatever it be, that is effective in gout, it is easy to understand that the local influence of the lesion itself will co-operate with that of its cause, and induce the progressive tendency that is a characteristic of this form.

DIAGNOSIS.—Myelitis is recognised by the rapid onset of symptoms indicating structural disease of the cord. Among these the failure of power is the most significant, although the subjective sensations that accompany it are [illegible] more obtrusive at the onset, and prevent mistake as to the meaning of the weakness. When they are absent the enfeeblement may be mistaken for mere prostration in cases in which some general illness coincides, and it is probable that slight myelitis sometimes occurs during an acute specific disease and altogether escapes detection. But among the early symptoms no one is more important, for its definite significance, than retention of urine; and its importance is increased by the fact that it may precede all symptoms in the legs and give a warning, the heed of which might sometimes enable lasting palsy to be prevented or life itself preserved. The diagnosis may be aided by the presence of such general symptoms as attend the occurrence of inflammation in other organs; but the absence of these is of little negative significance, while the presence of constitutional disturbance is as likely to mislead, as it is to suggest a local affection. The position of the myelitis must be inferred from the considerations already described. Its upper limit is indicated by the upper limit of the paralysis; that of sensation is most readily defined, and usually corresponds to that of motion. But the upper limit no more shows the extent of the disease below it, than the surface of water does its depth. The extent downwards must be gauged by the impairment of the functions of the cord as a central organ (reflex action and muscular excitability) in the parts paralysed, while the degree to which the various structures of the cord are damaged must be inferred from the character and degree of the symptoms in the affected parts. In ascertaining the state of the lower dorsal region the trunk and cremasteric reflexes are especially important.

Thus if called to a patient who has rapidly become paraplegic, the practitioner should first note the degree of motor and sensory paralysis of the legs (indicating whether the lesion is total or partial), and how high up the trunk the symptoms extend. He should then test the reflex action in the affected limbs and trunk, and ascertain the state of myotatic irritability in the limbs. These indicate the condition of the reflex arcs in the lower portion of the cord. Further information on this point is afforded by the state of muscular nutrition, and especially by the evidence of the state of nutrition of the nerve-fibres which is revealed by faradism. If reflex action is perfect, and the muscles have preserved their tone, this examination is a matter rather of scientific interest than of practical importance. It is useless to apply this test until five or seven days after the onset, because four or five days, and often eight or ten, elapse before the degenerative changes occur in the nerve-fibres. The examination may then be made without any risk, provided a very gentle current is employed, just sufficient to cause a contraction in the corresponding muscles of a healthy limb, and the current need not be applied to each spot for more than a second. The isolated faradic shock may be employed with advantage on account of the absence of the strong sensory stimulation that is caused by the current (see "Atrophic Paralysis"). If no change is found, the examination may be repeated at the end of ten days from the onset. If no muscles, at the end of that time, present any considerable diminution of contractility, it shows that no considerable nutritive change is taking place in the nerves, and that the grey matter from which the nerves proceed is not inflamed. On the other hand, if certain muscles present a diminution of irritability, others being normal, there is a focal lesion in the corresponding grey matter. If all the muscles of both legs present such a failure, there is inflammation of the lumbar grey matter, and if sensation is also lost, a total lumbar myelitis. So, if there is cervical myelitis with paralysis of all four limbs, the condition of irritability of the arm muscles shows whether the disease involves, or is above, the grey matter in the lower half of the cervical enlargement. If there is impairment of irritability, its extent in the two arms will afford an indication as to whether the myelitis is total, or whether it affects chiefly certain spots. The distinction from *simple polio-myelitis* depends on the fact that the symptoms are not purely motor. It is only when the myelitis is cervical or lumbar that the diagnosis is a matter of difficulty; in these cases the arms and legs are the seat of atrophic paralysis in ordinary myelitis, because the grey matter is involved. There is, indeed, polio-myelitis in such cases, but there is also more, and the impairment of sensation, together with the simple palsy of the legs when the disease is in the cervical region, shows the involvement of other parts than the anterior cornua,—which is the essential distinction. The most equivocal condition is that which exists when simple but severe polio-myelitis in the cervical region spreads to the white columns immediately contiguous, and causes weakness of the legs. But it never causes complete loss of power, even for a short time, and the legs soon recover, whereas in ordinary cervical myelitis the loss of power in the legs is often complete and prolonged, and is accompanied by impairment of sensation, not met with in polio-myelitis.

The distinction of the other varieties of myelitis rests on the special features already described. That of the *disseminated* form rests on the irregular distribution of the symptoms and the evidence of damage extending through a considerable vertical extent of the cord, but in some parts incomplete in degree. An inflammation which continues to extend after the first two or three days is certainly disseminated, and most subacute cases are of this variety, and so are those that are secondary to blood-states. The distinction is important, because this form is far more grave than any other, and more likely to cause death. It is doubtful whether *central* myelitis can be diagnosed during life; there is usually a slighter degree of myelitis in the other elements which causes symptoms resembling the ordinary form. There is, however, one exception to the rule that a spreading distinction is that in ascending myelitis sensation is affected, and if the patient survives there is a strong tendency to trophic disturbance in the skin, while many muscles waste and present loss of faradic irritability. In acute ascending paralysis, on the other hand, sensation is little impaired; some parts may be spared by the ascending palsy; bedsores do not form; and, if the case is not fatal, there is no change in the electric irritability of the muscles.

In *meningitis* the symptoms of irritation, severe pains, muscular rigidity, &c., are prominent, while they are absent in simple myelitis But in many cases the two conditions co-exist, and which disease is predominant can only be decided by the order and degree of the development of the symptoms. In *meningeal hæmorrhage* there is severe pain in the back and acute irritation of the nerve-roots.

The distinction from *multiple neuritis* has been considered in the account of that disease. It chiefly arises in the cases of polio-myelitis, presently to be considered; but a difficulty may exist when a parenchymatous myelitis affects the structures on which co-ordination depends, and produces the condition of myelitic ataxy. Such cases are distinguished from those of neuritic pseudo-tabes by the fact that some other structures are involved, so that symptoms are present that are only met with in diseases of the spinal cord. A very acute onset, so that the symptoms reach a high degree in a few hours, is also evidence of the myelitic origin. The case mentioned on p. 366 affords an illustration of these distinctions.

Myelitis is far from rare in patients of the age and sex in which hysteria especially prevails, and many cases are mistaken for *hysterical paraplegia*—sometimes because symptoms of hysteria concur, often merely because the patient is a girl and her legs well nourished. The converse error is very rare. The mistake occurs especially in cases of transverse dorsal myelitis, in which there is no wasting of the muscles of the legs. The gradual development of considerable excess of myotatic irritability is a symptom of great diagnostic value, and when this increases to characteristic extensor spasm there should be no room for doubt. If one leg is lifted from the bed, and the other moves with it owing to the rigid extensor spasm, organic disease is certain; hysterical contracture never fixes the legs to the pelvis so as to permit this effect. In hysterical paraplegia there may be retention of urine, but there is not incontinence. Among other conclusive symptoms of organic disease, a girdle pain, and incontinence of fæces, are of especial value. Trophic changes in the skin sometimes decide, even alone, the nature of the case.

PROGNOSIS.—The primary danger to life in myelitis depends upon the risk of respiratory palsy, and hence on the region of the cord in which the disease is situated. A disposition to spread is always of grave significance, and so also are indications of a serious blood-change, which is likely to increase the damage it has caused. On this account the prognosis is worse in disseminated than in simple transverse myelitis, and it is longer before confidence can be felt that arrest will endure. The risk is great whenever the cervical region is diseased, so that the intercostal muscles are paralysed, and in proportion to the nearness of the lesion to the origin of the phrenic nerve, on which life then depends. Hence a careful examination of the respiratory movements should be the first concern. Any indications that foci of inflammation have developed in the medulla oblongata are of extremely grave significance. Another danger is from acute trophic disturbances, and on this account the prognosis must be guarded whenever the lumbar enlargement is diseased, or is threatened by the extension of the inflammation. Hence bedsores, if forming early, within the first month, are an unfavorable indication; at a later period they have less influence on the prognosis. There may even be a phlegmonous condition of the viscera produced. The occurrence of cystitis, and especially of any indication of secondary kidney disease, increases very much the gravity of the case.

The prospect of recovery of power chiefly arises when the onset is over, and the disease has become stationary. Its degree depends (1) on the intensity of the disease, as shown especially by the loss of sensation as well as of motion, and (2) on its vertical extent as indicated by the impairment of the central functions of the cord; (3) on the early occurrence of symptoms of improvement. The longer loss of sensation continues, the less is likely to be the degree of ultimate recovery. (4) It is influenced to some extent by the cause of the myelitis, being better if this is due to a removable cause, such as pressure, than if spontaneous. (5) It is better when the disease is confined to the dorsal region than when rapid atrophy, &c., of the muscles show that the lumbar grey matter is involved. The longer motor palsy remains absolute, the less perfect will be the ultimate recovery. If some power returns within a fortnight, the amount that will be regained will probably be great; but even complete loss of power for six months does not preclude the ultimate return of the ability to walk, and I have even known this result after complete paralysis for a year from transverse myelitis. The development of increased myotatic irritability shows that improvement will not speedily occur, but even rigidity and the state of "spastic paraplegia" does not lessen materially the prospect of some recovery in a case in which the loss of power remains complete for a month or more. Indeed, the spasm often enables the patient to stand with a slighter degree of voluntary power than would suffice if the limbs were supple.

The fact of preceding syphilis must be allowed very little influence on the prognosis in a case of simple acute myelitis, but greatly improves the prognosis if there are indications that the inflammation is secondary to a more chronic process outside the cord.

Treatment.—If a case comes under observation at the earliest period, when only slight sensory disturbance and slight weakness of the legs indicate the commencing process, the question arises whether any treatment can avert the further development of the inflammation. If the symptoms are clearly due to exposure to cold, a hot bath, followed by free diaphoresis, should be employed, and followed by counter-irritation and the other measures now described. If considerable paralysis shows that the process of inflammation is fully developed, little can be expected from these measures, and it is better not to subject the patient to treatment that is incompatible with perfect rest. This, in all cases, is of paramount importance. Both functional excitation of the cord and movement of the spinal column should be avoided. The remarks regarding posture made in the account of the treatment of inflammation of the membranes apply also to that of the cord itself; it is most undesirable that the spine should be the lowest part of the body, and it is rather less difficult to keep the patient off the back in myelitis than in meningitis. A plank back-rest in the bed will be found a great assistance in securing comfortable rest on the side. If there is any reason to suspect hæmorrhage, or if there are indications of rapid extension of the inflammation, the prone position should be adopted, and even in myelitis it is well to adopt it at times as a change from the lateral posture.

The removal of blood from the skin of the back over the affected region, by leeches or wet cupping, is an old measure, which finds some theoretical justification in the fact that the blood from the structures behind the spine passes into the same veins as the blood from the spinal cord itself. Hence this measure may conceivably have some influence on the circulation in the cord. If the patient's strength is not such as to render the abstraction of blood desirable, dry cupping may be employed, or the vessels of the skin may be dilated by hot fomentations, or a mustard plaster, or hot water bags. By stimulating the cutaneous nerves, these agents may also influence, in a reflex manner, the vessels of the cord. The application of cold to the spine, as by a spinal ice-bag, has also been recommended. Contrary as these therapeutic agents seem, it is probable that each moderates local inflammation in the same manner, by causing first contraction and then dilatation of the vessels of the inflamed part, and so lessening the tendency to stasis of the blood, on which some effects of inflammation depend. Unless there is reason to suspect hæmorrhage, the application of warmth is the safer and probably, judging from experience, the more effectual. At the very onset of inflammation mild counter-irritation is unquestionably useful, and even a blister may be employed. The actual cautery of Paquelin, applied in a series of spots along each side of the spine, is probably more effective, and less liable to set up or intensify trophic disturbance; but when the process has reached a considerable degree it is very doubtful whether counter-irritation has much influence until the acute stage is over.

In other respects the treatment of acute inflammation of the cord must be guided rather by the nature of the process than by the character of the organ in which it occurs, and the fact that it is an acute local inflammation should be kept in view. A nutritious but unstimulating diet, aperients, and diuretics are desirable in all cases. If there is constipation a free purgative may be given. Whenever there is evidence of a morbid blood-state, it is important that this should, if possible, be improved, but we have still to learn how most toxæmic states can be neutralised. Nitrous ether may be given as a diuretic with some tincture of digitalis, which tends to render the circulation uniform, and to lessen stasis by its influence on the small arteries. The reason for diuresis is that probably no local inflammation occurs that is not associated with a morbid state of the blood, which may be to some extent relieved by the action of the kidneys. If any special drug is given, it may be combined with those above named. It is as difficult to ascertain the effect of the drugs which are supposed to exert a special influence on myelitis, as it is in the case of other local inflammations, which have no predetermined degree, and tend to subside when they have reached their height. Ergot was recommended by Brown-Séquard, chiefly on theoretical grounds, and has been extensively employed. In rare cases it has seemed to do good. In cases of hæmorrhagic myelitis it may reasonably be given with greater confidence, or ergotin (3 to 5 grs.) may be injected beneath the skin. Belladonna has also been recommended, but the evidence that it influences the morbid process is not strong. Mercury has been largely employed, given by the mouth and by inunction. The influence of mercury on the inflammation of internal organs does not seem so great as upon that of the fibrous tissues and of the structures that invest organs. Certainly in myelitis its effect is less distinct than it is in many cases of meningitis. Iodide of potassium seems to be no more efficacious than mercury. In cases of transverse myelitis occurring in syphilitic subjects the treatment for syphilis seems to have but little influence on the morbid process. It is true that such treatment is rarely adopted at the very onset of acute myelitis, but after the disease has developed energetic treatment does not seem to modify its subsequent course. This is true also of the subacute disseminated myelitis that occurs in the subjects of syphilis, and might be expected to be more amenable. I have known this form, concurring with syphilitic disease of the cerebral arteries, to develop and run its course to a fatal termination in spite of continuous antisyphilitic treatment, to which the arteritis yielded.

In the general management of a case of myelitis, two points are of extreme importance. One is to avoid, by scrupulous cleanliness and care, the exciting causes of bedsores. The skin should be most carefully watched, and any indication of deleterious pressure met by a change of position or an alteration of the mode of supporting the part. Cotton wool is very useful for this purpose. If there is a marked tendency to trophic changes the patient should be placed on a water-bed. When there is incontinence of urine, the difficulty of avoiding irritation of the skin is greatly increased. For males, a bed urinal is sometimes useful, but often it causes irritation and even sloughing of the prepuce, and then does more harm than good. A quantity of boracic or salicylic absorbent cotton wool, changed as often as it becomes saturated, is one of the best means of meeting this difficulty. It must be remembered that the prevention of bedsores is the prevention of one common cause of death. Should offensive sores form, a quantity of picked oakum, placed outside the immediate dressing, is a cheap and most effective means of preserving the air of the room from the fœtor of the sores, and is also a useful substitute for absorbent cotton wool, in the case of the poor, to absorb the urine or receive fæces that are passed unconsciously. Such material is also useful for relieving pressure when a water-bed cannot be obtained. The second point in management is the treatment of retention of urine. If there is either simple retention or overflow incontinence the bladder must be regularly emptied by the catheter, great care being exercised to prevent the introduction of contaminating germs. The importance of daily examining the abdomen to see that retention has not occurred, cannot be exaggerated. If the bladder is left full and the urine allowed to dribble away, inflammation is sure to be set up, and probably also pyelo-nephritis. If cystitis occurs, antiseptic washes must be used to lessen, as far as possible, the decomposition of the urine. Under this influence the cystitis usually lessens, and one grave danger to life is obviated.

When the disease of the cord has become stationary, the patient may be allowed to move, and a more tonic treatment may be adopted. Iron, quinine, or arsenic may be given. Strychnia must be given only in very small doses if there is any excess of reflex action. Occasional counter-irritation may be employed, repeated frequently if any improvement seems to result. The limbs may be rubbed, and any muscular wasting treated with electricity. It is not desirable to use electricity as a therapeutic agent while the cord disease is in an acutely active stage. There is no evidence that the application of electricity to the spinal column has any influence on the process of recovery of the cord. Its chief value is to maintain, as far as possible, the nutrition of any muscles of which the nerves have undergone degeneration. In cases of dorsal myelitis, in which the legs are well nourished, and the reflex action is excessive, it is better not to apply any form of electricity. The unavoidable stimulation of the sensory nerves tends to increase the reflex over-action. Careful attention should, in all cases, be paid to the position of the limbs during the stage of helplessness, so as to avoid as far as possible the development of contractions. For the condition of active spasm, which often develops after severe myelitis, not much can be done; such special treatment as can be adopted is described in the chapters on Primary Spastic Paraplegia.

ABSCESS OF THE SPINAL CORD.

Simple inflammation of the spinal cord scarcely ever goes on to the formation of pus, although leucocytes may accumulate at certain points of the grey matter so densely as to constitute microscopic collections of pus, and in very rare cases of this kind such minute abscesses have been sufficiently large to be visible to the naked eye. Pus only forms in the substance of the cord in considerable quantity in cases of purulent meningitis. In most instances the purulent meningitis has been of septic origin, rarely traumatic. In the former case suppuration within the brain may coincide with that in the spinal cord, and in the latter it may be very extensive, and occur at more than one spot. The symptoms are those of an acute irritative myelitis, but they are often lost in those of the purulent meningitis which precedes the disease of the cord itself. Their special feature is their association with a cause of septic suppuration, as well as with a high temperature and other symptoms of a septic blood-state.

A good example of the disease is a case recorded by Nothnagel. A patient suffering from cough and most offensive expectoration was suddenly seized with severe pains on both sides of the abdomen, attended by a sense of constriction and quickly followed by paralysis of the bladder and of the legs, with loss of sensation and of reflex action. An abscess of the cord was diagnosed. After death there was extensive purulent spinal meningitis, and the dorsal and lumbar cord contained an extensive collection of gangrenous offensive pus, which seemed to occupy the central part of the cord, from the cervical enlargement downwards. Some abscesses were found also in the white substance of the brain. In another case described by Ullman[*] two extensive foci of suppuration existed in the cord, one cervical, the other lumbar; the former had caused extensive destruction of tissue, and the pus had escaped into the subdural space. The affection was supposed to be secondary to gonorrhœa.

EMBOLISM OF THE SPINAL CORD.

The occurrence of embolism in the spinal cord has not yet been proved, but a few cases have been met with which suggest the possibility that the process has been the exciting cause of an acute myelitis. In a young man with mitral regurgitation, considerable weakness of the right leg came on suddenly—in a moment—with transient spasm. The onset indicated a sudden lesion in the cord, which might well have been the embolic obstruction of a small vessel. In a case recorded by Weiss, a boy aged sixteen, with chronic mitral disease, was suddenly seized with complete paraplegia, followed by bedsores, &c. He died four months after the onset, and the lumbar enlargement was found completely softened, with old coagula in the arteries. There was embolism of the kidneys and spleen, and the cortex of each cerebral hemisphere presented small foci of softening. Such cases justify a suspicion that embolism may be the cause of a sudden lesion of the cord in a patient in whom a source of embolism exists, and the process has occurred in other organs.

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[*] 'Zeitschr. f. kl. Med.,' xvi, 1889.

Chronic Myelitis.

The spinal cord may be the seat of chronic inflammation, which develops slowly, as such, in the course of a few or many months; and the condition may also occur as the sequel to acute myelitis, which, instead of subsiding, may persist, manifesting from time to time signs of activity. It is often difficult to say whether such a condition should be regarded as an acute myelitis that has not subsided, or a chronic inflammation beginning acutely. In many instances, indeed, it is probable that the disease is most accurately regarded as a combined form in which the causes of both are operative, and a subsiding acute inflammation is arrested and maintained by the influences that would be capable of inducing a primary chronic myelitis. Such progressive tendency is especially conspicuous in the disseminated form, in which the foci of inflammation may remain, and fresh ones may develop from time to time in a chronic or subacute manner.

The lesions of chronic myelitis resemble those of acute myelitis in seat and distribution, and intermediate cases connect them by analogous subacute forms. They differ from acute inflammation both in the longer time occupied in their development, and in the absence of the considerable vascular disturbance which forms part of the acute process. Such chronic myelitis may be focal, disseminated, or diffuse. In the former case it may involve the whole thickness of the cord at a certain level—*chronic transverse myelitis*; or only part of it, sometimes one half, occasionally for a considerable vertical extent. The *chronic disseminated myelitis* may resemble the corresponding subacute form in distribution, many points of inflammation being scattered through a small region, or through a large part of the cord, and its symptoms may become diffuse by the union of the effects of the disease.

The term "chronic myelitis" has, however, been used in a much wider sense, and has even been applied by some to all local processes attended by an increase of interstitial tissue, whether this is primary or is merely secondary to a degeneration of the nerve-elements. Thus the degeneration of the nerve-cells in progressive muscular atrophy has been regarded and described by many as a chronic myelitis of the grey substance, notwithstanding the fact that there is usually a degeneration of the whole motor path, in both segments, from the cortex of the brain, through the pyramidal tracts and motor nerves, and that the anterior cornua merely constitute, as it were, a focus in which the effects of the processes are especially conspicuous. Such a morbid process is evidently quite different in nature from a focus of ordinary inflammation, in which the primary change is interstitial and random in range and effects, and presents no limitation to function in the incidence of the consequences on the nerve-structures. Whenever this latter feature can be traced, the malady must be regarded as a "system disease," *i.e.* as a disease affecting a system of nerve-structures that have a common function. If "system diseases" are forms of inflammation, they are "parenchymatous inflammations;" and those that are chronic in course from beginning to end are more commonly termed "degenerations," and are so classed and described in these pages. Only, then, the chronic interstitial processes of random position and influence are here considered.

ETIOLOGY.—The causal relations of chronic myelitis, even as thus limited in conception, are very various. An inherited neuropathic tendency can sometimes be traced, but far more rarely than if the cases are included in which the nerve-elements suffer primarily. The disease is most common in early and middle adult life, but it occurs occasionally in old age; it is met with in both sexes, and is perhaps more prevalent in females in the first half of middle life and in males in the second—an important fact, because so many of the cases in young adult women are thought to be hysterical in nature.

Various conditions which lessen constitutional strength predispose to the disease. Definite causes often cannot be traçed, although many of the exciting causes of acute myelitis seem capable of giving rise to the chronic form, when acting for a considerable time, or frequently repeated, or in specially predisposed persons. Thus while a single intense exposure to cold may produce acute myelitis, frequent habitual exposure may cause the chronic form. Injury is a frequent cause, and seems to act in more than one way. Chronic inflammation may develop in the vicinity of a damaged spot, when the traumatic lesion may have been too slight to cause pronounced symptoms, or may have caused slight effects which are subsequently lost in the more severe and extensive consequences of the myelitis. In other cases the effects of the lesion may be too slight in degree to be at first perceptible, but (either by a slow influence on the processes of nutrition, or by extensive minute interstitial lesions that induce secondary traumatic inflammation) the symptoms of extensive damage to the cord slowly follow an injury that has no immediate effect on function. These cases will be further considered in a subsequent chapter. Repeated over-exertion has also seemed effective in some cases. Sexual excess has been thought to be a cause, but on less clear evidence.

Chronic myelitis may be secondary to other local morbid processes, especially to adjacent inflammation, which, when it also causes compression, invariably gives rise to chronic myelitis; the inflammation that results from pressure on the cord may be either chronic or acute.

Another series of causes consists of constitutional states capable of giving rise to local inflammations. Chronic alcoholism is, perhaps, the most influential and important of these. Chronic myelitis due to it is usually associated with chronic meningitis, and most intense at the periphery, beneath the inflamed membranes; but it thence extends into the substance of the cord, and sometimes tracts of irregular extent and position are involved, without connection with the membranes. This cause is one of especial importance, because it often co-operates with others, and determines effects that may seem to be independent. Its effect is met with, for example, in many cases of chronic myelitis that are excited by injury and seem to be due solely to the excitant, although the results of this would have been trifling and transient but for the tendency induced by the preceding and often profound effect of chronic alcoholism on the tissues. It also complicates many cases of multiple neuritis due to the same cause, in which the effects of the peripheral lesion obscure to a large extent those of the central mischief, by causing symptoms that prevent the recognition of the latter, since its consequences are lost, as it were, in those of the damage to the nerves.

The gouty diathesis is another cause of chronic myelitis. We are only beginning to realise how often it acts on many organs and tissues, and how frequently inflammation is thus induced. It is certain that this is a common cause of inflammation in the substance of organs and in mucous membranes, and chronic as well as acute myelitis is among its effects. Many cases are due to this influence, and it is instructive to note that, like chronic alcoholism, it is also a cause of inflammation of the peripheral nerves. The disseminated and progressive forms of chronic myelitis are especially due to it, and the analogy should be noted which these present to the irregular forms of perineuritis, of which this state is also a common cause. It is probable that many cases of chronic myelitis in young adults, at present mysterious in origin, and in the obstinately progressive character they sometimes manifest, will be found to be due to the influence of inherited gout, which is certainly capable of causing analogous inflammation elsewhere in the nervous system,—as, for instance, in the optic nerves.

Another cause of chronic myelitis is constitutional syphilis. The forms that are apparently due to this are of two types. (1) A diffuse interstitial inflammation, most intense at the periphery, with much new formation of tissue and thickening of the walls of the vessels, both processes extending into the cord from the pia mater, in which a similar damage exists. This form may be subchronic in course, and presents some histological correspondence to known syphilitic processes. (2) A focal very chronic indurating myelitis, such as shown in Fig. 106, which has no distinctively syphilitic feature, may occur in very late stages of the constitutional disease. In neither form does the influence of treatment afford evidence of the relation to syphilis. In the second, sclerotic variety it does not appear that treatment has any power to modify the morbid process, and in the first it is evident that any cicatricial process that could be induced would only maintain the damage to the nerve-elements, which would suffer further from the maintained or renewed compression under the prolonged contraction of the enclosing tissue. Yet the occurrence of these forms in the

FIG. 106.

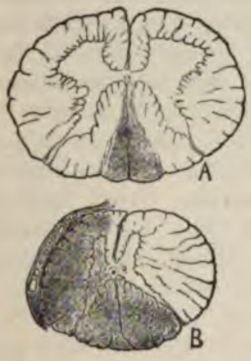

FIG. 107.

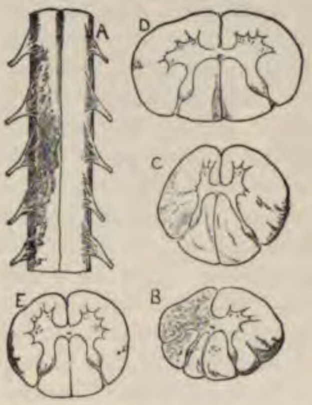

FIG. 106.—Chronic myelitis. (After Charcot and Gombault.) B, section at the level of the third dorsal, shows both the posterior columns and whole of left half inflamed, except the inner part of the anterior column. The affected part was grey and uniform in aspect, vascular, and firmer than normal. Pia mater and arachnoid thickened. A, section from cervical enlargement, showing secondary degeneration in the post.-med. columns. Similar foci existed in the pons, &c.*

FIG. 107.—Chronic sclerotic myelitis. (After Troisier.) A, posterior view of the affected part, lower dorsal region. The diseased areas were grey in aspect, and are indicated by shading. B, middle of lesion, showing extensive damage; C, 2½ cm. higher up, slighter damage; D, cervical region ascending degeneration, post.-med. cols., and a spot of disease in lateral column (probably part of the ascending ant.-lat. tract, secondarily degenerated). E, lower part of lesion. (See next figure.)†

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* The patient had had syphilis twenty-one years before. The spinal symptoms slowly developed during fifteen months, and consisted in paralysis with hyperæsthesia on the left side, with loss of sensibility without paralysis in the right, and a band of anæsthesia to pain around the thorax at the level of the lesion, but the mental dulness of the patient prevented accurate observation during the last few months of her life. (Charcot and Gombault, 'Arch. de Phys.,' 1873, vol. vi, p. 143.)

† The patient was a woman forty years of age. The symptoms slowly developed during the three years before death, and consisted of weakness with rigidity in the left leg, and impairment of sensibility in the right, first of the temperature sense and afterwards of touch. (No observations were made on sensibility for the last fifteen months of the patient's life, during which the lesion was slowly progressing.) (Troisier, 'Arch. de Physiologie,' 1873, vol. v, p. 716, Case 2.)

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few nerve-elements can be discovered, as in Fig. 108. In less affected parts of the white columns there is an irregular increase of the interstitial tissue, partly fibrillated, partly amorphous, or studded with nuclei (Fig. 108, upper small figure); while in other parts it may contain many nucleated cells, oval, fusiform, or stellate. The large stellate "spider cells" are sometimes very conspicuous, and interstitial fibrous tracts may be traced to their ramifications.

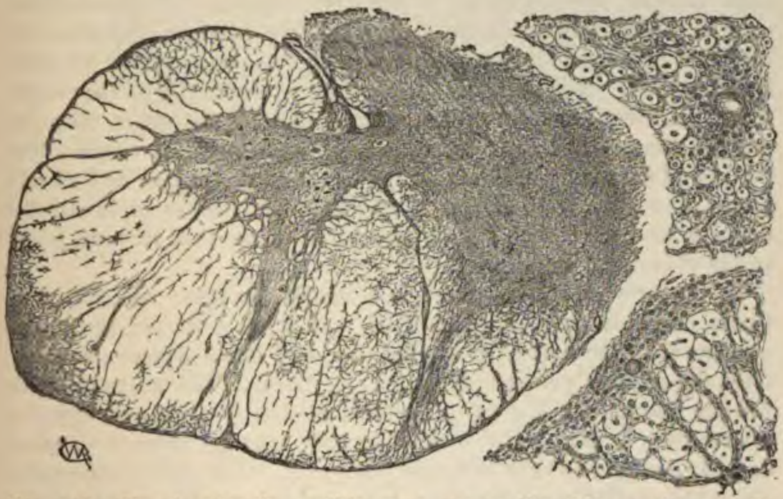

FIG. 108.—Chronic sclerotic myelitis, same case as Fig. 107; section near B, reversed. Almost the whole left half of the cord (right in the figure) is changed into a dense mass of shrunken connective tissue, and the right half is being invaded in many parts by the same process. The upper small figure is from the posterior column, showing a thickened vessel surrounded by nucleated tissue, among which lie nerve-fibres, many of them much smaller than normal, and some with swollen axis-cylinders. The lower small figure is from the right anterior column (left in figure); the pia mater is thickened and contains many nuclear elements; from it thick trabeculæ extend into the white substance and enclose spaces, from many of which the nerve-fibres have perished.*

The nerve-fibres waste before the interstitial process, or undergo destructive changes from the beginning. They may at first present the change by which their myelin stains more deeply than normal; afterwards they become narrowed, and the axis-cylinder is often irregularly enlarged. Ultimately many or all the fibres disappear from the affected area. Here and there the destruction of fibres may be out of proportion to the increase in the interstitial tissue, so that thickened and often granular trabeculæ enclose empty spaces. This is seen in the lower small illustration in Fig. 108; it is apparently the result of the participation of the nerve-fibres in the inflammatory process during a more general stage, in which all the elements of the tissue were involved. In the recent state, products of degeneration, granule corpuscles, &c., may be found abundantly in the altered

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* From a section by Prof. Pierret, kindly lent me by Dr. Dreschfeld.

[illegible] The walls of the vessels are thickened, and by this [illegible] the [illegible] of the arteries may be ultimately lessened; [illegible] smaller size may even be closed. Not only is the [illegible] thickened, but around them the increase of interstitial tissue is often greater than elsewhere, so that the appearance is [illegible] spread thence into the adjacent structures. Undue significance has been attached to this condition, especially in old patients [illegible] degeneration; it has been regarded as evidence that [illegible] disease sets up the myelitis, but it is a common appearance in [illegible] of chronic myelitis, and also in the later stages of acute inflammation. It is probably due to the greater activity of the process [illegible] immediate vicinity of the vessels, and to the share taken by [illegible] leucocytes in the formation of new interstitial tissue. The [illegible] arteries, moreover, occupy tracts of neuroglial and fibrous [illegible] proceeding from the pia mater, and the increase of interstitial [illegible] is connected with these; some of it is, on this account, found [illegible] vicinity of the arteries.

[illegible] the grey substance there is a similar increase of connective-[illegible] elements, so that the altered part takes a deeper stain with [illegible] than the normal grey substance, and has a denser structure, [illegible] the greater amount of supporting tissue. The nerve-elements [illegible] atrophied; the ganglion-cells, at first swollen and rounder, [illegible] shrink, until ultimately they may be reduced to small [illegible] bodies, and they even may disappear. The nuclear corpuscles [illegible] the central canal are often increased in quantity, and the canal [illegible], but this condition is so common in otherwise normal cords [illegible] importance can be attached to it. A considerable increase of [illegible] the central canal, closing it below and causing it to be [illegible] (Fig. 109, "syringomyelia"), has been ascribed to [illegible] inflammation around the canal, but the abnormal tissue is [illegible] of gliomatous nature, consisting of a development [illegible] is in congenital excess, owing to an arrest of develop-

[illegible] mater is greatly thickened over the inflamed parts, as [illegible] stated, and in some cases the affection of the membrane is [illegible] and that of the cord most intense beneath it—a condition [illegible] meningo-myelitis." It is this form that is shown in [illegible]. The tracts of new tissue that pass into the cord are dense [illegible]. As already mentioned, this change may be accompanied [illegible] and sometimes a more diffuse inflammation, as in A, Fig. [illegible] the affection of the cord is greater and that of the mem-[illegible] than in B. The chronic myelitis that occurs in syphilitic [illegible] present no specific characters. In some cases, however, [illegible] has been abundant, and has been associated with a [illegible] degeneration in the older portions of the lesion, and [illegible] degenerating hyperplastic inflammation of the pia [illegible] condition is, however, clearly one of chronic inflammation, and not of a syphilitic growth.* Nevertheless an actual growth does sometimes co-exist with a focus of syphilitic myelitis; it is probable that in such cases the growth is usually the primary change. This may be distinguished by the indications of compression and displacement of the adjacent elements of the cord, a condition which shows that there is an actual new formation.

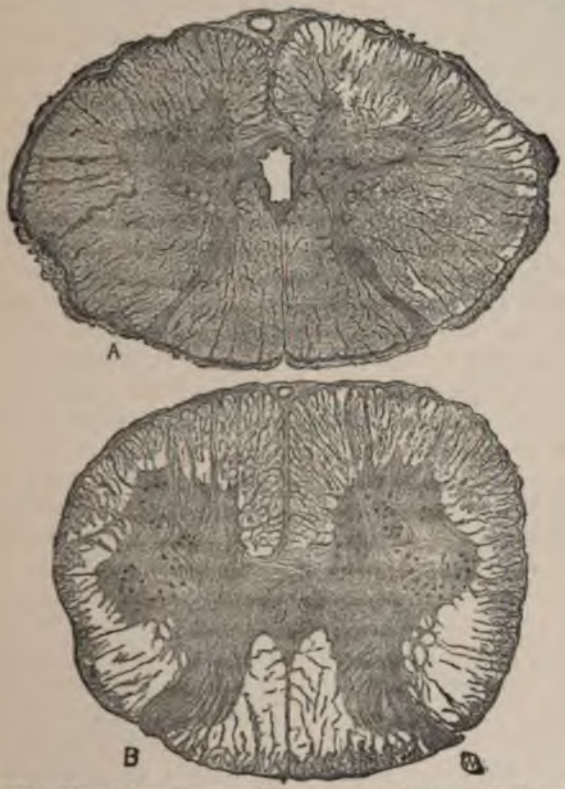

FIG. 109.—Chronic meningo-myelitis, due to alcoholism. Syringo-myelia, congenital, with central gliomatosis. A, lowest part of cervical enlargement; pia mater thickened, and from it tracts of tissue extend into the cord. The greater part of the section is the seat of a diffuse myelitis, in which the nerve-fibres could be seen with difficulty, and were separated by inflammatory products. The grey matter was also affected, and the nerve-cells much changed. The clear spaces in the right ant. cornu appeared occupied by products of degeneration. Central canal greatly dilated and surrounded by new growth, which in B (lumbar section) has obliterated the canal and thus caused its dilatation above. In this section the myelitis is chiefly peripheral, and the grey matter has, for the most part, escaped.

The more recent the inflammation, the larger is the proportion of cells and nuclei in the interstitial tissue; the longer it has existed, the greater is the amount of fibrillation it presents. The very chronic indurating forms are sometimes called "sclerotic myelitis," or "focal sclerosis of the cord." From the various areas of inflammation secondary degenerations proceed, ascending or descending, and these may complicate the aspect of the disease—a fact which has not always been sufficiently considered in interpreting the morbid appearances.

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* See Moxon, 'Guy's Hosp. Reports,' 1871, and Charcot and Gombault, 'Arch. de Physiologie,' 1873.

Symptoms.—The symptoms of chronic myelitis vary much according to the character of the morbid process. For the most part they correspond in character to those that are produced by acute inflammation, differing, however, in their mode of onset. It is therefore not necessary to repeat them in detail. Different cases may manifest almost every symptom that can be produced by disease of the spinal cord There may be various motor and sensory paralyses, contractures, and even muscular atrophy, but their common feature is their gradual, and often successive development. In the most common form, focal myelitis of the dorsal region, partial or transverse, the symptoms are paraplegic. They differ but little whether there is a single area of disease, or whether several foci near together implicate adjacent structures. Motor power is usually impaired far more than sensibility. Subjective sensations are often prominent and early symptoms, whether there is anæsthesia or not,—tingling, "pins and needles" in the legs, a sensation as if fur or wool covered the skin, sometimes with dull pain in the legs and back, especially after exertion, and commonly also with a well-marked girdle pain at the level of the chief lesion. Some loss of sensibility, partial in kind or in seat, will often be found if carefully searched for, but there is not often absolute loss of sensation except in the rare cases of intense local sclerotic myelitis, by which conduction may be arrested as completely as by a growth. The onset of the motor weakness is gradual; many months may pass before the power of walking is considerably impaired. As the legs get weak, excess of reflex action is developed (unless the reflex structures are damaged). The knee-jerk is increased; foot-clonus occurs when the legs are in certain positions; and a tendency to spasm gradually develops, and increases to the condition typical of "spastic paraplegia." The sphincters frequently share the impairment of voluntary power. Co-ordination of movement is sometimes affected, usually manifested as general unsteadiness, and defect of equilibrium, rather than as pure ataxy.

The symptoms may come on simultaneously in the two legs, or one may be affected before the other, and occasionally the paralysis reaches a high degree in one leg while the other still possesses fair strength. When one half of the cord is affected almost alone (as in the lesion shown in Fig. 109), there may even be a crossed paralysis of motion and sensation.

Chronic polio-myelitis is often regarded as a distinct variety, but much confusion has arisen from the fact. If chronic myelitis develops in the cervical and lumbar enlargement, muscular wasting is usually added to the other symptoms. Similar atrophy occurs also in the widely diffused form of chronic myelitis, in which almost all parts of the cord suffer, and it may, indeed, be the most conspicuous symptom. It is always irregular in distribution in these cases, and it may be combined with other symptoms, palsy and anæsthesia, also irregular in extent and seat. The muscular wasting varies in the rapidity of the development, according to the character of the inflammation, and there are corresponding variations in the relations to it of the loss of power in the muscles, and also in the electrical reactions they present. In very chronic cases there is a slow failure of electrical irritability to both currents; in others of more rapid course the voltaic irritability persists longer than the faradic irritability, and there may be a distinct degenerative reaction. The symptoms may commence in the arms or in the legs, but they ultimately become general in most cases, and, according to the place of their commencement, may seem to have an ascending or descending course. Occasionally the disease begins in the dorsal region, and after a time, perhaps years, it spreads to the enlargements, and the muscular wasting is added.

The course of chronic myelitis is usually slow and progressive. The symptoms often increase so gradually as only to attain a considerable degree at the end of two or three years. At any stage the progress of the disease may cease. The chronic course may be varied by occasional more rapid increase of the symptoms, due to subacute or even acute processes in the diseased parts. Every degree of chronicity is met with, and subchronic cases, in which the symptoms develop in the course of a few months, effect a gradation to the acute and subacute forms of myelitis. The duration of the disease varies from six months to ten years or more. Thus in one case the symptoms slowly increased during about seven years, and then became stationary, and the patient died twenty-three years after the onset.

Diagnosis.—The diagnosis of chronic myelitis rests on the slow development of symptoms indicating a random process in the spinal cord, *i. e.* a process which damages irregularly structures of various functions, and thus cannot be looked on as a "system disease." The maladies from which it has to be distinguished differ according to the seat and character of the inflammation. Dorsal transverse or focal myelitis may be confounded with compression of the cord, with a tumour in it, and with primary lateral sclerosis. The distinction from pressure rests on the absence of a cause of compression, such as bone disease, —especially the absence of the signs of a morbid process outside the cord, preceding those of damage to the cord itself, *e. g.* the pains that indicate irritation of nerve-roots. A tumour within the spinal cord also causes, in most cases, more irritation of the root-fibres than does chronic myelitis. If myelitis involves one half of the cord much more than the other, the symptoms may closely resemble those of an intra-medullary growth. But a tumour never causes much damage to one half of the cord without interfering considerably with the functions of the other half, and the symptoms in chronic myelitis may be strictly unilateral. The distinction from primary spastic paraplegia, the so-called "primary lateral sclerosis," is often one of some difficulty. The motor state of the legs may be identical in the two diseases; in each there is the same extensor spasm, and in each there is a slow, gradual, and apparently simultaneous onset of the weakness and spasm. The distinction is that in primary spastic paraplegia the symptoms are purely motor; there is no indication that the lesion extends beyond the motor structures. In chronic myelitis, on the other hand, there is such indication, either by the impairment of sensation, or by the presence of a girdle pain. This distinction is sufficient in the majority of cases. The distinction from insular sclerosis is in many cases difficult. By Oppenheim and others, indeed, it is asserted that most cases described as chronic myelitis are really cases of disseminated sclerosis. The presence of cerebral or bulbar symptoms would naturally suggest such a diagnosis in a given case. But in insular sclerosis any very obvious affection of sensation is unusual.

The form that involves the grey matter has to be distinguished chiefly from pachymeningitis and degenerative muscular atrophy. In pachymeningitis the muscular wasting may be similar, but anæsthesia is usually much more considerable in range and in degree, and there is more pain in the limbs. If there are similar symptoms in both arms and legs, myelitis is far more probable than pachymeningitis, since the chronic inflammation of the membranes is less extensive than that of the cord. From progressive muscular atrophy, the chief difference is in the random distribution of the wasting, the absence of symmetry, and indications of irregular damage to other structures in the cord, as the occurrence of pain and of loss of sensation. But it must be remembered that, by some authorities, most cases of chronic spinal muscular atrophy are regarded as due to chronic myelitis.

For the distinction from other less common diseases, the reader must be referred to the description of them. Like all other organic diseases, cases without wasting of the muscles, occurring in females, are often regarded as "hysterical paraplegia;" the distinction rests on the points already described. It is, however, one of the diseases in which the least excusable errors in diagnosis occur, and in which their effects are often the most disastrous.

PROGNOSIS.—Chronic myelitis is a very grave disease, on account of the intractability of the morbid process, its frequently progressive tendency, and the persistence of damage which is slowly produced. But cases vary widely, especially in the two former features, and it is a disease in which general prognostic rules can scarcely be formulated. The observed tendency of an individual case is alone a trustworthy guide. Arrest of the disease is often obtained, but actual recovery is rare. The prognosis is little affected by the seat or extent of the process, except that the implication of the grey matter of the enlargements is generally an unfavorable indication. The severity of the lesion, as shown by the degree of the symptoms, is significant chiefly in cases of long duration, and is subordinate to the course and mode of onset. If it can be arrested, the prospect of improvement is better in proportion to the slowness with which the disease has developed and the shortness of the time the symptoms have lasted. In general, moreover, the prospect is better, the fewer the foci of inflammation, in males than in females, and in cases of uniform than in those of recurrent or relapsing course. Preceding syphilis does not materially modify the prognosis; hence the great importance of the diagnosis from syphilitic growth, in which suitable treatment has a most certain effect. The ultimate danger to life is least in focal myelitis in the dorsal region; it is greatest in the disseminated form, especially in the less chronic cases with a tendency to sloughing of the skin, and in the more chronic forms with muscular wasting. The indications drawn from the invasion of the respiratory muscles, and from the presence of any of the complications that so often terminate life, are the same as in other diseases of the cord.

TREATMENT.—The first and most important measure is the improvement of the general health, by rest, change of air, and tonics. All causes of physical and mental depression must, as far as possible, be removed. Over-exertion, and even fatigue, should be avoided, and the patient should be kept, as far as possible, from exposure to cold. Absolute rest for a short time is often useful at the outset of the treatment, especially when there has been a somewhat rapid development of symptoms. In cases of purely chronic course absolute rest should not be maintained for more than a few weeks, or the patient may find it hard to regain his former muscular power. Counter-irritation at the situation of the disease is often useful, and most so in cases in which there is spinal pain or tenderness. Repeated sinapisms or blisters may be employed, but a mild form of the actual cautery is on the whole the most useful. A superficial burn or even slight vesication is sufficient on each side of the spine opposite the affected part, and it is best effected by means of the Paquelin cautery. It should be repeated as soon as the skin has healed, and if linear cauterisation is adopted the fresh line can be made beside the old one. A hot douche to the back, at a temperature of 103° or 104° F., applied daily, has been strongly recommended by Brown-Séquard. Warm brine baths, and various thermal mineral waters, have been also said to do good. A sea voyage is often of service, combining as it does the maximum of fresh air with the minimum necessity for exertion. Drugs, unfortunately, often fail to influence the morbid process, but nevertheless they are occasionally useful, so as distinctly to justify their careful trial in most cases; and it is important to remember that those that are useless in one stage or period of the disease may yet be of unquestionable service at another stage. Tonics, as quinine and iron, should be given if indicated. Most of the measures recommended, and precautions advised, in the treatment of acute myelitis are needed in the chronic form, and it is therefore superfluous to repeat them. Those drugs that most deserve a trial are arsenic, small doses of mercury (such as $\frac{1}{24}$ gr. of the red iodide), and iodide of iron. Energetic mercurial treatment rarely does good, even when the patient has had syphilis, and sometimes it does harm. Iodide of potassium seems to have little influence on the disease. Nitrate of silver, ergot, and phosphorus have been recommended. Strychnia is chiefly useful in cases in which there is muscular wasting, but is of far less value than in the degenerative muscular atrophy. The treatment of other symptoms is that suitable in primary spastic paraplegia or progressive muscular atrophy, and described in the account of those diseases.

COMPRESSION OF THE SPINAL CORD.

Compression of the spinal cord is a common consequence of various morbid processes. Inflammation is almost always excited by the pressure, and interference with function occurs, partly from the pressure and partly from the resulting myelitis. The symptoms produced have, in different cases, many characters in common, although they vary according to the mechanism of the compression and the acuteness of the inflammation. It is only when there are indications that an acute myelitis has damaged the nerve-elements that we are justified in regarding the interference with function as an effect of the inflammation.

CAUSES.—The morbid processes that may compress the cord are those that involve an encroachment on, or occupy part of, the vertebral canal. The chief are the following:—Disease of the bones of the spinal column, especially caries; growths in the spine; aneurism eroding the bones and then compressing the cord; growths in the membranes; thickening of the dura mater. These processes have usually only a small vertical extent, and the pressure they exert rarely extends over more than a few inches.

PATHOLOGICAL ANATOMY.—The cord usually presents evidence of the compression it has endured in considerable narrowing at the spot, where it may be indented and flattened, or cylindrical. Sometimes the reduction in size is extreme; for an inch or so the cord may be reduced to one third of its normal diameter, and it has even been found no thicker than a crow-quill. An example of flattening is shown in Fig. 110. On the other hand, there is sometimes very little narrowing to be discerned. At the compressed part the cord is usually red in tint if the compression is recent, but grey if it has lasted for some time; its consistence is lessened in early cases, and increased in those of long duration. The change in colour and consistence is due to the inflammation of the substance of the cord which always results from pressure, and may often be traced for some distance above and below the compressed part. When there is much compression there is always much inflammation, but considerable myelitis may occur when the amount of compression is slight. The inflammation may be chronic and slow, developing in proportion to the pressure, or it may be subacute or acute, even when the pressure is gradual. The signs of inflammation are very distinct on microscopical examination, and resemble those in other forms of myelitis. There is a general increase in the interstitial tissue; in this, at first, various cell-forms may be found, but it ultimately presents the appearance of a dense reticulum. The nerve-elements undergo degeneration, and abundant masses of myelin, granule corpuscles, and corpora amylacea are visible in the fresh state or in glycerine preparations of the hardened cord (Fig. 111, c). Many nerve-fibres persist, however, with a narrowed sheath of

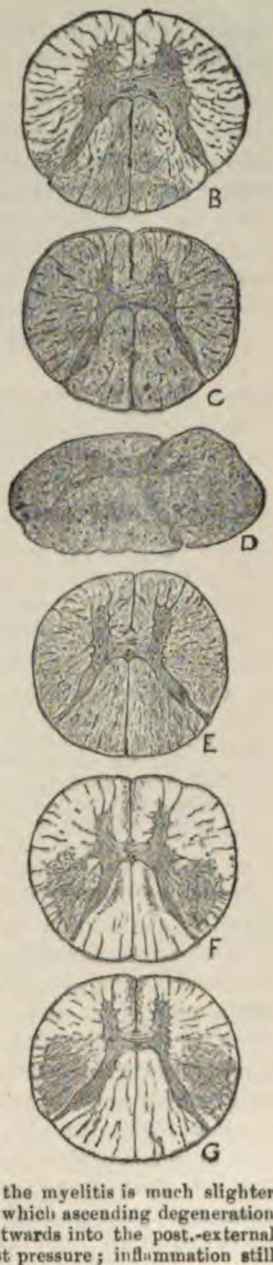

FIG. 110.—Compression of the spinal cord and pressure-myelitis, in a case of caries of the spine. D, mid-dorsal region, near the chief point of greatest compression; the cord is narrowed from before backwards, and is uniformly damaged by inflammation, so that the grey substance can scarcely be distinguished. C, 1½ inches higher up, shows a slighter degree of myelitis, still extending through the whole thickness of the cord. B, first dorsal; the myelitis is much slighter and the chief disease is in the posterior columns, in which ascending degeneration occupies the post.-median columns and extends outwards into the post.-external columns. E, 1½ inches below the point of greatest pressure; inflammation still extending through all the elements of the cord. F, 2 inches lower down, shows only descending degeneration in the pyramidal tracts, anterior and lateral, and also the "comma-shaped" descending degeneration in the anterior part of the post.-external column. In G, at the lowest part of the dorsal region, the comma-shaped degeneration has ceased, and only that of the pyramidal tracts remains. (From preparations by Dr. F. G. Penrose.)

myelin, and it is probable that these regain the power of conducting, in spite of the persistence of considerable, and even dense sclerosis in the part. In extreme cases all the fibres seem to be destroyed at the point of chief compression, but there is never an actual division of the cord itself. The grey matter can scarcely be distinguished from the white, even with the microscope, and the ganglion-cells become shrunken and atrophied. The walls of the small vessels are often thickened by spindle-cells, disposed more or less concentrically to the cavity, which is encroached upon and may be obliterated (Fig. 111, A, B), a process that must add to the degree of damage to the cord. The signs of inflammation gradually lessen above and below the compressed part, but often extend for some inches in each direction. Beyond its limits the usual ascending and descending degenerations may be traced (Fig. 110). The nerve-roots passing by the seat of compression suffer from the pressure, and from interstitial inflammation excited in them. They are usually grey in tint and ultimately waste, and may even be reduced to fibrous threads. The microscope shows increase of the interstitial tissue, wasting of many nerve-fibres (Fig. 111, D), and enlargement of some axis-cylinders.

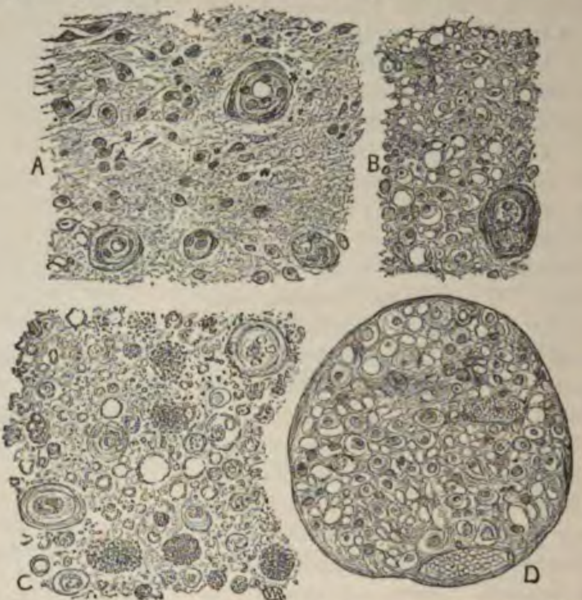

FIG. 111.—Pressure-myelitis; portions of the section D in Fig. 110 more highly magnified. A, from the grey matter, numerous angular and fusiform cells; vessels with walls thickened by a growth of cells, narrowing the cavity, which in some is obliterated. B, from the white columns; thickening of the interstitial tissue, nerve-fibres in part destroyed, in part narrowed; a vessel with thickened walls. C, glycerine preparation from white substance; abundant products of degeneration of the nerve-fibres, in part aggregated into granule masses. D, section of a nerve-root passing by compressed area; increase of interstitial tissue, many nerve-fibres narrowed, some with swollen axis-cylinders.

SYMPTOMS.—The effects vary much according to the degree of pressure, its rapidity, its direction, the amount and character of the inflammation produced, the amount of damage to the nerve-roots, and the position of the disease. The symptoms in most cases enable the fact of slow compression to be inferred, even when there is no indication of the cause. They are of two classes: (1) interference with the function of the nerve-roots at the level of the morbid process; (2) interference with the functions of the cord itself. The cord symptoms consist chiefly in impaired conduction, manifested in the parts below the lesion. The central functions of the cord (reflex action, &c.) at the level of the lesion are abolished by the pressure, but the symptoms of this abolition are often lost in those of the damage to the nerve-roots.

Of the root symptoms the most constant is pain, extending along the course of the nerve-fibres, and through the area of their distribution. The seat of these pains depends on the position of the disease; they may be felt in the arms, around the thorax or abdomen, or in the legs. They are usually sharp pains, resembling neuralgia in character, sometimes accompanied by tender points. Occasionally, when felt in the limbs, they are referred especially to the joints. The pain may be intermitting or constant. It may even be absent, as in a case of aneurism of the arch of the aorta already referred to. It may exist alone for a long time in a disease that increases slowly, and sometimes may be the only symptom, although some compression of the cord occurs. With it there is usually hyperæsthesia of the skin, often intense. After a time anæsthesia develops in irregular areas, in spite of the persistence of the pain,—the condition termed "*anæsthesia dolorosa.*" Irritation of the motor nerve-roots may cause painful contracture of the muscles, but this is, on the whole, rare, and the chief motor symptoms are due to the interruption of the fibres—muscular weakness, gradual in onset, and accompanied by wasting. The rapidity of the atrophy varies much, and with it the electrical reaction. When slow, there may be merely a progressive diminution in irritability to faradism and voltaism; when rapid, there is often the degenerative reaction, and sometimes the "mixed form," from the partial damage to the fibres that supply a muscle.

These local root symptoms are usually the first, and to them are added, after a time, the indications of interference with the function of the cord itself. There is weakness in the parts supplied from the cord below the lesion. The loss of power is usually gradual, but sometimes comes on rapidly, even in the course of a few hours, when an acute myelitis is set up by the compression. With the weakness there is a marked and early increase in the superficial reflex action, more constantly conspicuous than in most other diseases of the cord, and therefore of some diagnostic importance. The myotatic irritability is also increased, a foot-clonus being almost always present. Pains in the legs may occur, even when the disease is above the lumbar enlargement, usually dull and aching. There is often formication and tingling. There may be no impairment of sensibility in the parts below the lesion, even when there is complete motor palsy; in many cases, however, there is some sensory loss, complete only when the spinal lesion is very severe. Conduction of painful impressions may be delayed, sometimes for thirty or even forty seconds. When the pressure is lateral, one leg may be first and most affected, but the other side is usually soon involved, since neither the mechanical effect nor the inflammation remain limited to one side of the cord. The sphincters are often affected. The tendency to the formation of bed-sores is rarely great, unless the lumbar enlargement is compressed or myelitis descends into it.

The course of the symptoms varies according to that of their cause. When an acute myelitis has been set up, improvement may occur for a time even if the cause of pressure is slowly progressive. This is true also of root symptoms when they are rapidly developed. If the compression ceases to increase, the cord may recover its conducting function, although it remains narrowed. Sensation is often regained in the legs when motion remains absent, but both motion and sensation may be regained although the narrowing of the cord persists. It has even been found no larger than a goose-quill at the compressed spot, although the paraplegia had passed away. In such a case many narrowed nerve-fibres are found in the section of the compressed portion, and it is probable that many axis-cylinders persist, with an envelope of myelin so narrow as to be recognised with difficulty, but which suffices for their conducting function. Under slow pressure the axis-cylinders may not suffer interruption, although the myelin disappears. Regeneration of axis-cylinders, as in the peripheral nerves, is also possible. In some cases, especially when a considerable myelitis has been set up, the motor path may recover, but the posterior columns remain affected, so that power returns, but without co-ordination,—"secondary ataxy" resulting.

The DIAGNOSIS of slow compression rests on the root and cord symptoms already described, and especially on their coincidence with a cause of compression. Of the former, the sensory symptoms are the most characteristic. In many cases other indications of the compressing disease assist the diagnosis. These vary in character; the most frequent is considerable local tenderness of the spine. If root symptoms are absent the diagnosis is much more difficult, unless the morbid process manifests itself externally. If there is a suspicion of compression in a case of slow paraplegia, an early and considerable excess of superficial reflex action gives additional weight to the suspicion. Even without any other symptoms to indicate compression, this is suggested by symptoms which begin on one side and gradually spread to the other.

The chief disease from which compression of the spinal cord has to be distinguished is a subacute or subchronic transverse myelitis.

Besides the indications of a cause of compression already mentioned, the chief distinction depends on the presence of symptoms indicating irritation of the nerve-roots, and especially on the fact that these symptoms precede those of the lesion of the spinal cord itself. Although a very chronic myelitis is occasionally attended by much irritation of the nerve-roots, the symptoms of this succeed, instead of preceding, the damage to the cord, and the course of chronic myelitis is often much slower than is that of most forms of slow compression. The distinction from a growth within the spinal cord rests chiefly on the early occurrence of impairment of the central and conducting functions of the cord, and also on the partial distribution, and often slower course, of the symptoms. The effects of an intra-medullary growth vary widely in different cases, and if there is an early irritation of root-fibres, the distinction may have to depend on the presence or absence of the signs of an external compressing process.

The diagnosis of the cause of compression is often much more difficult than that of its occurrence The facts that the patient is in the first half of life, and that he inherits a tubercular tendency, suggest caries. Slightness of root symptoms is also in favour of caries or a tumour within the spinal cord; the presence of these symptoms does not, however, render caries less likely, unless the pain is extremely severe and is greatly increased by movement,—characters that should always suggest a growth in the bones of the spine. Root symptoms extending over a considerable vertical area suggest pachymeningitis. Additional details of the differential diagnosis will be found in the account of the several compressing diseases.

Prognosis and Treatment.—The prognosis depends on the cause, and the amenability of this to treatment. As a general rule, however, the more rapidly the cord symptoms develop, the more chance there is of improvement, because the greater share does inflammation take in their production. The treatment is that of the cause of the compression, and that of myelitis.

ANTERIOR POLIO-MYELITIS; ATROPHIC SPINAL PARALYSIS.

In certain diseases of the spinal cord, wasting of the muscles is a prominent symptom. In these the lesion involves, exclusively or in part, the anterior grey cornua, and the muscular atrophy depends on the destruction of the nerve-cells, from which the motor nerve-fibres proceed, and on the consequent degeneration of the fibres. The muscular wasting does not depend on the nature of the lesion; it occurs equally whether the nerve-cells are destroyed by hæmorrhage, by inflammation, acute or chronic, by slow degeneration, or by pressure. But the rapidity with which the destruction occurs influences the character of the symptom. Time is required for the change in the nutrition of the muscles; one or two weeks elapse, even in the most acute cases, before wasting is distinct. The nerve-cells also form part of the voluntary motor path, and their disease at once interrupts this; hence, in acute cases, paralysis is rapidly developed, and the atrophy succeeds it after an interval. In chronic lesions, on the other hand, the changes in the cells and muscles progress so slowly that the weakness and wasting appear to come on together. The wasting may even seem to precede the weakness, but a careful examination will show that power is defective as soon as there is distinct atrophy. It is customary to separate the two groups by terming those in which the palsy precedes wasting, "atrophic spinal paralysis;" and those in which wasting is apparently simultaneous with weakness, "spinal muscular atrophy."

The atrophic paralyses are due to an acute or subacute process in the anterior cornua. The lesion is probably, in most cases, inflammatory in character, a cornual myelitis, or "polio-myelitis" (Kussmaul). In some cases, which differ chiefly in the suddenness of the onset, it is probable that the lesion is hæmorrhage and not inflammation.

The chronic atrophies are due to a slow degeneration of the cells and fibres. Between the two forms, however, we meet with others of intermediate course, subacute or subchronic, due to processes of corresponding character,—usually inflammation.

ACUTE ATROPHIC PARALYSIS; ACUTE ANTERIOR POLIO-MYELITIS.

Infantile Paralysis; Essential Paralysis of Children.

Acute atrophic paralysis is a disease in which voluntary power is lost in the course of a few hours or days, and in which some of the paralysed muscles undergo rapid wasting while others recover. The onset of the palsy is often preceded or accompanied by indications of general disturbance. The muscles which waste usually remain weak, and contraction of their opponents may lead to permanent distortion of the paralysed limbs. Although it is usually a form of acute myelitis, its characters and course are sufficiently special to make its separate consideration desirable. The disease is the most common form of paralysis in young children, and has hence received the name of "infantile paralysis." Before anything was known of the lesion on which it depends, it received (from Rilliet and Barthez) the unmeaning designation of the "essential paralysis of children," a term that is falling into merited disuse. The spinal lesion was first demonstrated by Prevost (1865), and soon afterwards by Lockhart Clarke and by Charcot and Joffroy. Its constancy has been abundantly proved by numerous observations during the last thirty years. The occurrence of similar symptoms in adults was noted by Vogt (1858) and Duchenne (1864).

ETIOLOGY.—The disease occurs at all ages, but is ten times as frequent in the first decade as in all the rest of life. It is, moreover, especially a disease of later infancy. Of the cases in early life, three fifths occur in the first three years of life, nearly one fifth in each year. The numbers in each successive year (of 214* cases of which I have notes) were—in the first 54, the second 58, third 39, fourth 17, fifth 23, sixth 8, seventh 4, eighth 6, ninth 5. Of the cases that begin during the first year, nearly all begin in the second six months; the earliest of the series occurred at two months, but one at the twelfth day is described by Duchenne. The friends often date the disease from birth, but there is no real evidence of its intra-uterine development. After childhood it is most frequent between ten and twenty, and very rare over forty. One case, however, occurred at sixty-three. Many supposed subacute cases in adults have been examples of multiple neuritis.

In childhood the two sexes suffer nearly equally (males 133, females 103, in 236 cases), but after ten the malady is almost confined to the male sex. The influence of heredity is small, but a family tendency to suffer may now and then be traced; two and even three brothers or sisters have been affected (Seeligmüller, &c.). One of my own patients had a sister, and another two cousins, affected with the same disease. When more members of a family than one are affected, they usually suffer simultaneously. The inherited tendency may be indirect; of a patient in whom the disease came on in adult life, two brothers had suffered from hemiplegia and an uncle from paraplegia.

The disease presents a remarkable relation to season, occurring far more frequently in summer than in winter. Sinkler, who first called attention to the fact, found that in Philadelphia four fifths of the cases commenced in the five hot months, May to September. In this country the relation is equally apparent; three quarters of the cases occur during the hottest third of the year, June to September. The distribution of 160 cases was as follows:—3 occurred in January, 5 in February, in April 2, May 3, June 22, July 32, August 37, September 25, October 15, November 8, December 8. In many cases the weather was very hot at the time; in some, however, the disease came on in cooler weather after a hot period. It would therefore seem that the heat generally acts as a remote cause, and is only occasionally concerned directly in the exciting mechanism. The fact is the more important because cold has long been regarded as the most potent exciting cause. That exposure to cold is an occasional cause is certain, although the number of cases in which it can be traced is not large; and it is usually a general and not a local exposure, and has not often been immediately preceded by exposure to heat. Exceptions are chiefly met with in adults. Thus one patient, aged sixteen, when perspiring from a long ride after the hounds on a hot day in September, lay down on a sofa beneath an open window from which a draught blew on his back. Two days afterwards the first symptoms commenced. In another case the onset followed a day on a steamer in cold, wet weather, preceded by much paddling in the sea during extreme heat. I have several times known the disease to be due to sitting on damp grass. Two or three days is the interval which usually elapses between the exposure to cold and the onset of the symptoms, but great variations are met with, and the constitutional disturbance may commence within twenty-four hours of the exposure.

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* Of these 214 cases 116 were seen and analysed by Sir W. Gowers, and 98 by Dr. James Taylor. They include both hospital and private cases.

Over-exertion in walking seems occasionally to aid in exciting the disease; in some cases the child has walked far more than was proper the day before the onset, and in others habitual over-exertion has been permitted. A traumatic influence, such as a fall, occasionally precedes the onset in such a way as to make its influence probable. Thus, in one severe case, a boy eight years old was thrown over the head of a donkey, and the onset occurred five days afterwards. A girl of four fell off a chair, striking her head, and was stunned for a moment; two days later she was sick and feverish; the temperature (101°) continued raised till the sixth day, when general loss of power was found, and the residual state was atrophic palsy of both legs and the right deltoid. The cases following falls and over-exertion present the same relation to season as do the rest.

There is no proof of any relation between the disease and the process of dentition. The period of dentition coincides with the rapid functional development of the nervous system which succeeds its structural development in the first few months of life. Moreover this period is often one of deterioration of health, in consequence of changes of diet and other causes. The disease occurs before and after the period of teething, and it exhibits no increase in frequency at the period of the second dentition.

Many children are perfectly well at the time when the disease occurs. A few are in conspicuously defective health. Thus in one case the child had been rendered feeble by long-continued diarrhœa. I have only once seen the disease in the subject of inherited syphilis. It has been thought to be occasionally secondary to acute febrile diseases, but the frequency of the relation has been unquestionably overrated; in some supposed cases in adults there has been multiple neuritis only, and in children initial general disturbance is constantly mistaken for an independent general disease, and the opinion is often maintained after the discovery of the paralysis, which is then supposed to be of secondary origin. Thus in one case initial pyrexia, headache, and vomiting were supposed to indicate scarlet fever. Paralysis of all four limbs came on, with difficulty in swallowing, and the latter was supposed to render certain the diagnosis of scarlet fever, although there was no sore throat or rash. In older children and adults the disease is often thought to be secondary to rheumatic fever, in consequence of the rheumatoid character of the pains. The error is the more likely to occur if the symptoms follow exposure to cold. It is conceivable that catarrhal affections may be produced by the exposure to cold which induces the disease; but of the 214 cases referred to above, not one was related to any acute specific disease, or to rheumatic fever. Chronic alcoholism has been thought to be a cause in adults, but it is probable that most cases supposed to be due to this influence have been instances of multiple neuritis.

SYMPTOMS.—The general characters of the symptoms have been already mentioned—an acute onset, often with constitutional disturbance; paralysis, at first wide-spread, afterwards passing away, except from a region in which the muscles rapidly atrophy, and in which, although partial recovery may occur, more or less weakness and wasting persist.

The general disturbance, which is apparently due to a morbid blood-state, may be severe or slight. In most cases there is pyrexia, with its usual accompaniments, headache, prostration, loss of appetite, restlessness. Vomiting is very common, and often causes the illness to be mistaken for stomach disturbance. Occasionally there is also diarrhœa. These symptoms of general disturbance usually last for a few days, sometimes for only a few hours, and they are occasionally, but not often, absent. The palsy develops in most cases in the course of this disturbance. A common history is for a child to seem ill and feverish, to be put to bed, and the next morning to be found to have lost power. In other cases the febrile disturbance lasts for several days before the paralysis comes on. But the relation of the general disturbance to the onset of the paralysis presents great variations, which appear (from a comparison of cases) to be independent of any variations in, or special features of, the spinal symptoms. The constitutional symptoms occasionally succeed the onset. Thus a child, well one day, was found next morning to have both legs paralysed, and was feverish and sick; the general disturbance continued for a week. The elevation of temperature is usually moderate in degree; an example of its character, in what may be regarded as a fairly typical case, is that of a child whose temperature on the second day of illness was 101·2°; on the third, 101°; the fourth (on which the palsy developed), 100°; the fifth, 99·5°; the sixth, 98°. It is usually, as in this case, highest at the commencement, and sometimes reaches 103°, 104°, or even in rare cases 105°. The constitutional symptoms are present in most of the cases in which a fall appears to excite the disease, and are of the usual character; they are as marked in the cases that appear to be spontaneous as in those that follow exposure to cold.

Convulsions occasionally attend the onset, chiefly in young children. In some instances, moreover, the initial symptoms are those of severe cerebral disturbance. Thus one child had repeated general convulsions during thirty hours, and afterwards was in a state of deep coma for thirty-six hours, the temperature at the onset being 105°. This was succeeded by slighter pyrexia and prostration for three weeks, and it was only at the end of that time that general loss of power was found to have come on. The arms recovered, but atrophic palsy persisted in the legs. In another case, a child of seven months became ill, febrile (102·8°) and comatose with pin-point pupils; it remained in that state for four or five days, and then gradually recovered, but with general powerlessness, rigidity of the legs, and tenderness of legs and back. The rigidity and pain passed away, but the right deltoid and, to a less degree, the biceps and triceps, and the whole of the right leg remained paralysed, and presented characteristic atrophy. In adults there is sometimes marked somnolence before the onset, and occasionally slight cerebral symptoms, such as diplopia, giddiness, or (very seldom) delirium. A medical student found, one day, that he saw double; on the next day he felt giddy, and became feverish (102°); he slept for forty-eight hours, and on the fourth day found his right arm was weak; atrophy of the deltoid, &c., followed. The cerebral symptoms usually pass away together with the indications of general disturbance, and hence are probably due to the blood-state, but occasionally slight facial weakness has persisted for several weeks. In rare cases the onset is accompanied by symptoms of slight transient spinal meningitis, which must be regarded as a coincident effect of the cause, since the membranes may be affected early. Thus a child, after being feverish and vomiting for two days, became universally rigid, so that strychnine poisoning was thought of. The rigidity passed away two days later, when the right arm and leg were found to be paralysed. Another child presented all the symptoms of spinal meningitis a week after the onset of paralysis of the arm, and as they passed off the leg also was found affected. When the general illness is severe, the paralysis is often not discovered for some time, especially in a child; the immobility of paralysis is often mistaken for the inertia of prostration.

In older children and adults another common initial symptom is the pain already mentioned, sometimes referred to the muscles, sometimes to the limbs generally, occasionally to the course of the nerves. It is often absent, but may be very severe and accompanied by extreme tenderness of the limbs. In such cases the pain may be supposed to be in the joints, because it is produced by moving them, when it is really in the nerves, which are mechanically disturbed by the movement of the joints. In these cases there is neuritis, which may come on at the same time as the palsy, although independent of it. The joints themselves may be the seat of pain, and, when the exposure to cold has been severe, inflammation of one or more joints has been known to come on soon after the onset. Pain may be felt also in the back, usually dull and aching. It may be severe, especially when there are symptoms of spinal meningitis. Instead of being initial, pain in the limbs may succeed the onset of paralysis, and it occasionally continues for several weeks, especially when seated in the nerve-trunks. In some cases in which the onset and course are characterised by severe pains, the motor effects have entirely disappeared in course of time, indicating that the condition was one of neuritis. Tingling or formication in the limbs may be described by older patients, but sensation is seldom affected, even temporarily.

The paralysis is almost always rapidly developed. It varies much in its initial range. Only part of a limb may be affected, or there may be universal loss of power; but in the majority of cases the paralysis is intermediate between these extremes. Two or three limbs are affected—both arms, both legs, or the legs and one arm. When all four limbs are paralysed the neck muscles also may be weak, and even swallowing may be impaired. The other muscles supplied by the cranial nerves escape, as a rule, and the sphincters are seldom involved. Occasionally there is retention of urine, sometimes incontinence, chiefly in severe cases, and it may then last a long time. For instance, a child aged two and a half years woke up one morning with headache, fever, and weakness of the legs, which rapidly increased to complete paralysis. Four days later the arms also became weak, and in a day or two more the urine escaped involuntarily. The arms began to recover in six weeks, and were well in six months: both legs wasted and remained permanently paralysed; the incontinence of urine lasted for a year. In another case, with incontinence for a few weeks, the residual palsy was in the lower legs. As in these instances, the symptom is met with chiefly when the lumbar region is diseased.

The paralysis may commence in one limb and quickly spread, reaching its maximum extent in a few hours to a few days, but very often the actual onset is so rapid or so escapes careful observation in consequence of the general illness that all the parts first affected are found to be paralysed when it is discovered. Occasionally the onset is in two distinct stages, separated by a few days; sometimes the loss of power develops slowly in the course of one or even two weeks, in cases that must be regarded as "subacute," but do not differ in other respects from the ordinary form. Examples of the common onset have been already given; as another, may be mentioned the case of a boy, aged a year and four months, who one day seemed ill, was sick, and was put to bed. The next day he could scarcely stand alone; on the following day he could neither move the legs nor sit up in bed. In about ten days the left leg began to improve, but there was enduring paralysis and wasting of the whole of the right, and of the lower part of the left leg. In another case, a child seemed ill, and was kept in bed for five days; on the third day it was noticed that the left arm was not moved so much as the right, and by the fifth day the arm was quite powerless; at the end of a fortnight improvement commenced. In the adult, the mode of onset presents nearly the same characters. A lady, twenty-five years of age, sat down for some time on wet grass.

Two days later general rheumatic pains came on, which were very severe in the legs on the following day, and the legs were distinctly weak. In the course of the next forty-eight hours the arms also became feeble, and at the end of a week she could scarcely move her arms, and her legs were absolutely paralysed. In a fortnight improvement commenced, first in the arms and then in the feet; but the hip and thigh muscles remained paralysed and rapidly wasted.

Cases are sometimes seen similar in other respects to those now under consideration, but in which the paralysis comes on suddenly, and without general disturbance. Thus a child, aged two, was walking along, when he suddenly could not stand, and fell down on his knees; both legs were powerless. Slow improvement followed in the left leg, and wasting in the right. The suddenness of the onset makes it probable that the lesion is hæmorrhage, and not simple inflammation. Spinal hæmorrhage is known to occur in early childhood. Hence, while we must class these with the other acute spinal atrophies, we are not justified in regarding them as cases of polio-myelitis, since we do not know whether the hæmorrhage is secondary to commencing inflammation or is primary. The former is probable, because the two sets of cases occur under the same general conditions of onset, and hence they cannot be separated. Moreover, constitutional disturbance is more often absent in cases of actually sudden onset than in others, although it is sometimes considerable. Thus a child had been ill for two weeks, when, being able to walk about, it suddenly fell to the ground; the left leg was found powerless, and the right weak. It is probable that a sudden onset is more frequent than is suggested by the facts that can be ascertained, since the palsy often comes on during the night or during rest; for instance, in one case a child sat on a chair for an hour, when the left leg was found powerless.

In some cases of sudden onset the initial palsy is universal, and in such cases as the following it is probable that a limited hæmorrhage in the cervical region causes rapid pressure on all the adjacent structures, which afterwards recover from the effects of the pressure. A girl, aged seventeen, suddenly felt tingling in the left hand. In a few minutes the whole arm was powerless, and then the right arm became paralysed. She had a strange sensation at the back of the neck, and went upstairs; as she was going up, her legs became weak, first the left and then the right. In less than half an hour from the time of the first symptom, there was absolute universal paralysis, with difficulty in breathing and in swallowing. Improvement (in the opposite order to the onset) commenced the same evening and was complete in about six weeks, with the exception of the muscles of the left forearm and hand, which rapidly wasted and were permanently paralysed and atrophied. Another girl, while walking across a road, suddenly felt a "sort of shock" as if someone had given her a knock between the shoulders. She became giddy, and instantly felt tingling in both arms, especially in the right, which became weak before she had got to the other side of the road. Ultimately the arms recovered, but permanent paralysis and wasting of the intrinsic muscles of each hand were left.

The initial loss of power is as a rule far more extensive and severe than the permanent affection; in a small number of cases the two correspond. But the extent of the early palsy, that of the enduring atrophy, and the relation between the two, vary extremely, so that no general or even common laws can be discovered. Indeed, in all features of the disease variability prevails to such an extent as to baffle attempts to describe them in general terms, and a better conception of the malady can be gained from such illustrative instances as are here given than from general descriptions. One general fact is unfortunately true of almost all cases, namely, that the palsy scarcely ever passes away entirely, and that where wasting occurs, wasting remains.

After the paralysis has reached its height and ceased to increase or spread, whether the point is attained suddenly or rapidly or slowly, it remains stationary for a time, which varies from a few days to six weeks, and then lessens. The improvement occurs first in the parts last affected, and gradually spreads until, usually at the end of from one to three months, all parts have recovered except those which are to be permanently affected. In these the muscles are toneless and flaccid from the first, and in two or three weeks there is distinct wasting, which rapidly increases, until the shape of the limb is changed, and, in extreme cases, scarcely any of the volume of the muscle can ultimately be detected. In fat children the appearance of the limb may be less altered, and it seems that, in some of these, an interstitial growth of fat makes up for the diminution of muscular tissue. In older children and adults the muscles are often tender to the touch during the process of wasting.

When the atrophy is distinct the muscles no longer contract to faradism, and, if the motor nerves are tested, they also will be found to have lost irritability. The change depends on degeneration of the nerves, and the muscles usually present the "degenerative reaction" in its characteristic form. The loss of faradic irritability is distinct, in severe cases, as early as the end of the first week, and even by the fifth or sixth day. In a patient, for instance, who is universally, and apparently uniformly, paralysed, one or more groups of muscles may be found to have lost irritability, and in these we know that there will be lasting paralysis and wasting, while the other parts will recover. In a severe case, in which the muscles most affected atrophy completely, the loss of faradic irritability may be permanent. The voltaic irritability remains, increased in degree, for two, three, four, or six months, then slowly falls, as the muscular fibres themselves degenerate, and ultimately, at the end of one or two years, it disappears. It may, however, persist for a long time, if the fibres are stimulated by electricity,—even for three or four years, although the paralysis remains absolute. In rare cases, electrical irritability quickly disappears, even to the voltaic current, from the destructive degeneration of the muscular fibres. More commonly, after six or twelve months, some faradic irritability returns. It may be slight, due to the recovery of a few fibres, insufficient in number to restore any bulk or power, but the new fibres which recover do so perfectly, so that the irritability becomes normal in degree, although the contraction which can be thus produced is very slight in amount. In other cases considerable recovery occurs, so that some power and volume are regained, although the muscles remain below the normal size. In the muscles which are weak but do not waste there is no degenerative reaction, although there may be slight diminution of excitability to each current.

The paralysis, as a rule, is motor only. Sensation is impaired only in extremely rare cases (about 1 in 50), in which inflammation in the lumbar region is so intense as temporarily to impair all the conducting functions of the cord. Thus, in a girl aged five, in whom the permanent state was a symmetrical atrophy of all the muscles below the knees, with a slighter affection of the thigh-muscles and flexors of the hips, the initial general loss of power in the legs was accompanied, for a few days, by complete loss of sensation below the knees. In the similar but more severe case of a boy, aged four, in whom the lasting palsy was general throughout both legs, the anæsthesia continued for two months. In many cases there is a history of temporarily disturbed sensation attending the onset, probably the result of œdema around the inflammatory area. When sensation is lost there is always incontinence of urine.

Reflex action necessarily ceases in the parts related to the affected muscles. That from the skin is at first lost where there is weakness, but it returns with or soon after the recovery of power in the less affected parts. Where there is persistent paralysis it remains absent. The myotatic irritability is lost in the same or even greater degree. For instance, no knee-jerk can be obtained if the extensors of the knee are affected even in slight degree. The loss depends on the interruption of the muscle-reflex centre by the disease in the grey matter. In rare cases of severe cervical polio-myelitis, the inflammation spreads beyond the grey matter to the lateral white columns; there is wasting of the arms, but paralysis without wasting in the legs, and in the latter the myotatic irritability may be increased above the normal, so that the foot-clonus may be obtained. In the course of a few months, however, the condition of the legs becomes normal.

A common effect of the disease is to retard the growth of the bones in the affected limb, so that these gradually become shorter than their fellows, and the difference in size increases with the amount of general growth. Hence, it is greater, the younger the patient at the onset of the disease. If one part of a limb is more affected than another, the corresponding bone suffers most in growth. The effect is sometimes considerable, when the atrophy is slight. Seeligmüller has described the opposite effect—an actual elongation of the bones, attributed to the fact that the growing epiphyses suffer traction instead of the normal compression, but this is extremely rare.

Those joints that depend for their support on tendons which pass over them become lax when the muscles are paralysed, and the articular surfaces may be no longer kept together. Thus when the deltoid is wasted the head of the humerus may fall out of the glenoid cavity. The head of the femur may fall from its proper position in the acetabulum, and actual luxation may occur when the paralysis of the muscles is unequal, and the less affected unopposed muscles undergo the secondary contracture immediately to be described. That of the abductors and rotators may cause infra-pubic dislocation, and that of the adductors iliac dislocation.* I have known the patellar tendon to be greatly elongated, from the unopposed action of the extensors of the knee, so that their contraction caused the patella to rise three inches above its normal position.†

During the chronic stage of the disease there is great tendency to the occurrence of permanent shortening of muscles, with consequent displacement of the parts to which they are attached. Grave deformities are thus produced. It occurs only when the several muscles acting on a joint are unequally paralysed; the less affected muscles are no longer subject to the normal extension by their opponents, and hence gradually become contracted, and the contraction is fixed by tissue changes in them, so that they cannot be passively extended. It may occur in muscles which are unaffected, and it is uncertain how far it is favoured by a slight degree of paralysis. Posture also aids the production of those muscular contractions, and so also in the case of the foot does the relative shortening of the limb.

Bedsores are almost unknown, even in the acute stage of the disease. A slight local elevation of temperature in the most affected parts has been observed in the early stage, but subsequently the affected limb is colder than the other. This is due, in part at least, to the want of the aid to the circulation which is, in health, supplied by the muscular action.

The persistent paralysis may occupy the whole or part of one or more limbs. The legs suffer three times as frequently as the arms; the respective percentage being 21 and 68. It is less frequent for both legs or for one arm to suffer, and still rarer combinations are those of both legs and one arm, of both arms, of the arm on one side and the leg on the other, while the rarest of all is the affection of leg and arm on the same side. The limbs on the left side of the body are more often permanently affected than those on the right; the difference is greater in the leg than in the arm. The left leg is paralysed alone twice as often as the right.

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* See Karewski, 'Archiv f. klin. Chir.,' Bd. xxxvii.

† In a case shown me by Mr. Heather Bigg.

When the paralysis of a limb is incomplete, the part involved varies much in different cases, and as different parts of two or more limbs may be affected, the combinations of palsy which result are extremely varied. The grouping of adjacent muscles is sometimes distinctly that of functional association; often it is random; but the most frequent condition is to have an irregular affection of the muscles that are associated in the centre. While the grouping is that of function, some of the muscles are only slightly affected, and others suffer with them that are not functionally associated. In the legs, the paralysis, in very severe and fortunately very rare cases, may be absolute, involving all the muscles of both limbs. Usually it is partial, and then the muscles below the knee suffer more often than those above the knee. The calf muscles are affected less frequently or in less degree than the muscles in front of the tibia or the peronei. Hence talipes equinus is a common deformity, and its occurrence is aided by the shortening of the limb, in consequence of which the foot has to be extended to bring the ball to the ground. Either the tibialis anticus or the peronei may be more affected, with the result, in the former case, of talipes valgus, in the latter of talipes varus. In the case shown in Fig. 112 the peronei have suffered most in the left leg and least in the right, producing an unsymmetrical but

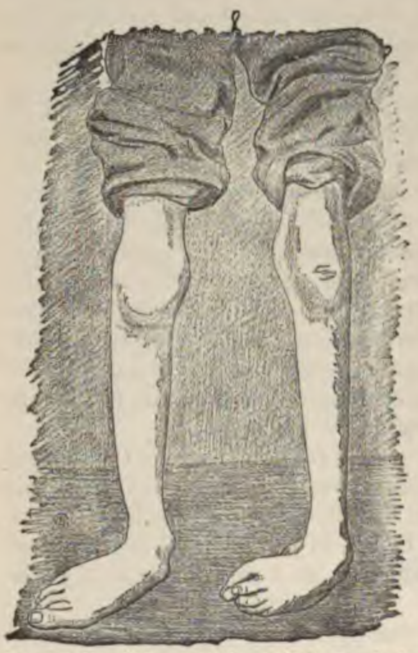

FIG. 112.—Old atrophic spinal paralysis. Talipes valgus in the right leg from paralysis of the anterior tibial; t. varus in the left, from paralysis of the peronei.

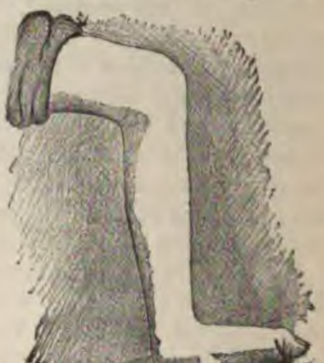

FIG. 113.—Atrophic spinal paralysis, affecting chiefly the calf muscles, with resulting contraction of the flexors of the foot, causing talipes calcaneus.

corresponding distortion of the feet. This affords an illustration of another fact, namely, that when there is a partial affection of both legs the paralysis may be unsymmetrical in the two. Much less commonly the calf muscles suffer more than the others, and there results talipes calcaneus, as in the case shown in Fig. 113, and the intrinsic muscles of the foot often suffer with those of the leg. In the thigh, the extensors of the knee are affected more frequently than the flexors, and hence flexor contraction is very common, and may even cause subluxation of the joint. The flexors of the hip often suffer, generally with the extensors of the knee. Less commonly the glutei are involved, but never alone.

In the arm, almost all the muscles are sometimes affected, as in the case shown in Fig. 114, but all are seldom entirely paralysed. The intrinsic muscles of the hand often suffer; either the thenar muscles or the interossei may be most damaged. An instance of such paralysis of the interossei is shown in Fig. 115. The forearm muscles are frequently affected, but the supinators may escape when the other muscles are involved. The deltoid suffers more frequently than any other single muscle of the arm. It may be paralysed alone or in association with other muscles; occasionally the deltoid, supra- and infra-spinatus, biceps, and supinators are all involved in the "upper arm type" of palsy of Erb* (see p. 111). But other muscles than these may be

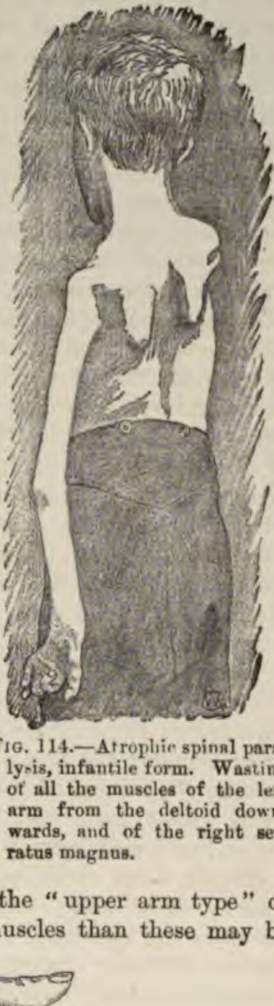

FIG. 114.—Atrophic spinal paralysis, infantile form. Wasting of all the muscles of the left arm from the deltoid downwards, and of the right serratus magnus.

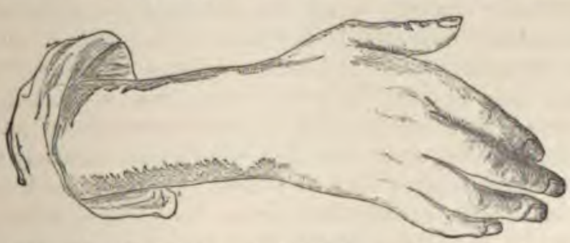

FIG. 115.—Atrophic spinal paralysis (adult form) affecting the interossei and thenar muscles, and also the deltoid.

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* See a paper by Dr. Beevor, 'Med.-Chir. Trans.,' 1885.

associated with the deltoid. The irregularity of the grouping is shown by the fact that the triceps is often affected with these muscles. In the case shown in Fig. 115 the deltoid and intrinsic muscles of the hand were wasted, and no other muscles. The serratus magnus is occasionally affected as in Fig. 114; in this case, although the left arm had suffered severely, as the figure shows, on the right side only the serratus was involved. With the serratus, the upper part of the pectoralis major is sometimes associated in paralysis (as it is in normal function, see p. 37), the lower part being unaffected; it was so in this patient. The middle part of the trapezius and other scapular muscles are occasionally involved. The neck muscles rarely suffer, but the diaphragm is occasionally paralysed. Although the intercostals and other trunk muscles are so often weakened in the early stage, considerable permanent atrophy is very rare. Curvature of the spine is sometimes produced in consequence of the patient being allowed to sit up while the muscles are weak. It may be a lateral or an antero-posterior curve, according as the weakness is on one or both sides. I have met with considerable depression of the lower part of the wall of the thorax on the left side from paralysis of the intercostal muscles at the spot, perhaps combined with hindered growth of the ribs. The muscles supplied by cranial nerves are scarcely ever affected. I have once seen complete paralysis of one side of the face, associated with wasting in the limbs, in an otherwise characteristic case, and an instance of affection of the face and tongue has been recorded by Pasteur.*

The *Course* of the disease has been already sketched. There are (1) an initial stage of paralysis, rapidly increasing, often accompanied with fever; the duration of this stage is from a few hours to a week or even more; (2) a stationary period, which lasts for a week to a month; (3) a stage of "regression," during which the palsy passes away, except from certain parts in which wasting occurs; this regressive period usually occupies from one to six months; (4) a chronic stage (overlapping the last), during which atrophy continues; slight improvement may occur, but contractures and deformities are developed. The duration of this stage is indefinite, because, wherever muscular tissue remains and some voluntary power returns, this slowly improves, by increased development of the muscle under the influence of use, and this continues in slight degree for years. This gain is often, however, more than counterbalanced by the interference with growth, and by the occurrence of deformities. Even in the slightest cases complete recovery is extremely rare, and the limb remains smaller than its fellow. On the other hand, death from the disease is equally uncommon, and occurs chiefly in the early stage. It is not improbable that some children die from the initial disturbance before its nature is recognised. Now and then death occurs at the end of the first week or ten days, from universal paralysis or from some such severe cerebral disturbance as existed in the cases mentioned above, or as followed the spinal lesion in a case in which paralysis in the arms came on upon the third day of the initial fever and reached its height on the fifth day, faradic irritability being lost by the seventh in many muscles of the left arm; a little rigidity of the neck then occurred, and on the tenth day the patient began to vomit, complained of increased headache, and had hallucinations; on the eleventh day he became comatose and died. Bronchial catarrh is an occasional cause of death, when the respiratory muscles are weak.

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* 'Lancet,' 1887, vol. ii. It is probable that when the cranial nerve nuclei are affected, the vital centres become involved in most instances, so that the patients do not survive.

Relapses are very rare, and occur chiefly in the form of a separate later extension of the disease. Second attacks are almost unknown; one instance only was met with in the series of 214 cases.

Sequelæ, beyond those mentioned as part of the symptoms of the disease, are also very rare. As a rule the general and nervous health of the patients, and the duration of life, are uninfluenced by the local palsy. In a few cases, however, some other chronic affection of the spinal cord has come on when the subjects of infantile paralysis have reached adult life. Progressive muscular atrophy has been several times observed to start from the paralysed limb, and even to be apparently excited by a fracture of the bone.* Acute and subacute polio-myelitis in adult life have also been observed as a sequel, and I have twice seen the symptoms of lateral sclerosis slowly developed, in one case at seventeen, in the other at twenty-eight. In the spinal cord of the subject of old infantile paralysis there seems thus to persist some disposition, slight though it be, to fresh disease, and the cases of lateral sclerosis mentioned show that the liability to disease is not limited, as has been thought, to the grey matter.† The altered structure of the bones has been known to facilitate their fracture; in one case the bones in the affected limb were broken on three separate occasions (Dutil).

PATHOLOGICAL ANATOMY.—For a long time, infantile paralysis was believed to be a peripheral affection, having its seat in the muscles. But improved methods of examining the nerve-centres have shown changes in the spinal cord which have the characters of an acute inflammation of the grey substance of the anterior horns. The correspondence between the infantile and adult forms makes it certain that the latter are, in most instances, of the same nature. Subacute atrophic paralysis in adults has been found, however, to depend on disease of the nerves in some forms that were thought to be spinal, but in these the palsy is strictly symmetrical, and when it is irregular in distribution the disease is certainly identical with the infantile form.

Observations on the changes in the spinal cord in the early stage

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* Raymond, 'Prog. Méd.,' 1889; Dutil, 'Gaz. Méd. de Paris,' 1881.

† Compare Ballet and Dutil, 'Revue de Médecine,' January, 1884, p. 18.

are few. A case recorded by Dr. D. Drummond, in all p
one of this disease, is by far the earliest on record, and is
earliest possible.* A child, five years old, died after a f
acute illness. The spinal cord, in the region of the fourth
cervical nerves, presented undue redness of the anterior gr
The vessels running from the surface to the cornu were
with blood. The microscope showed distension of capill
minute extravasati
grey substance, s
the neuroglial elem
of the ganglion-ce
were granular with
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early observation is
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116). In this an
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advanced than in
mond's case. They a
widely spread in sli
gree, and attain consi
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The microscope shows
vasated blood often
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c), and scattered thro
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litis. There are also
corpuscles and other p
of degeneration of the
elements. These el
and especially the
nerve-cells, have almost entirely disappeared. Where the ch
slighter in degree there is a leucocytal infiltration, a few
corpuscles are seen, while the nerve-cells may be structurally

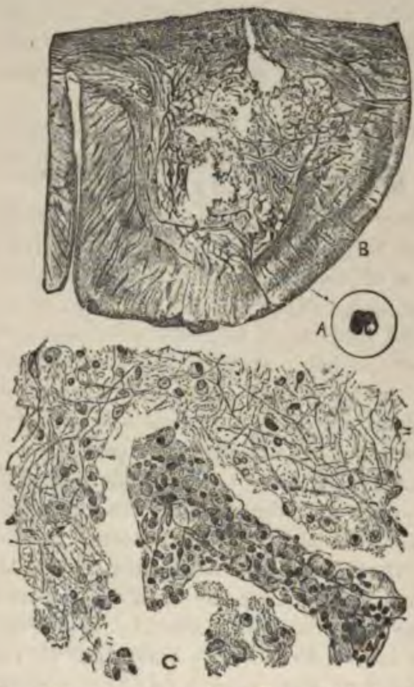

FIG. 116.—Acute anterior polio-myelitis in a child 2½ years old, six weeks after the onset. (After Charlewood Turner.) A. Section through the lowest part of the lumbar enlargement, showing a cavity visible to the naked eye, on the left side. B. Left anterior half of the cord under a low magnifying power, showing destruction of almost the whole anterior cornu. C. Portion of anterior cornu in the upper part of the lumbar enlargement. Numerous corpuscles lie in the granular protoplasm and reticulum of fine fibres. A small artery is encrusted with several layers of corpuscles and nuclei.

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* 'Brain,' April, 1885.

† 'Path. Trans.,' vol. xxv, p. 20

but swollen and granular. The morbid appearances may be confined to the anterior cornua, or may extend in slighter degree into the adjacent lateral column, in which the nerve-fibres near the grey matter may undergo destructive degeneration, and other signs of

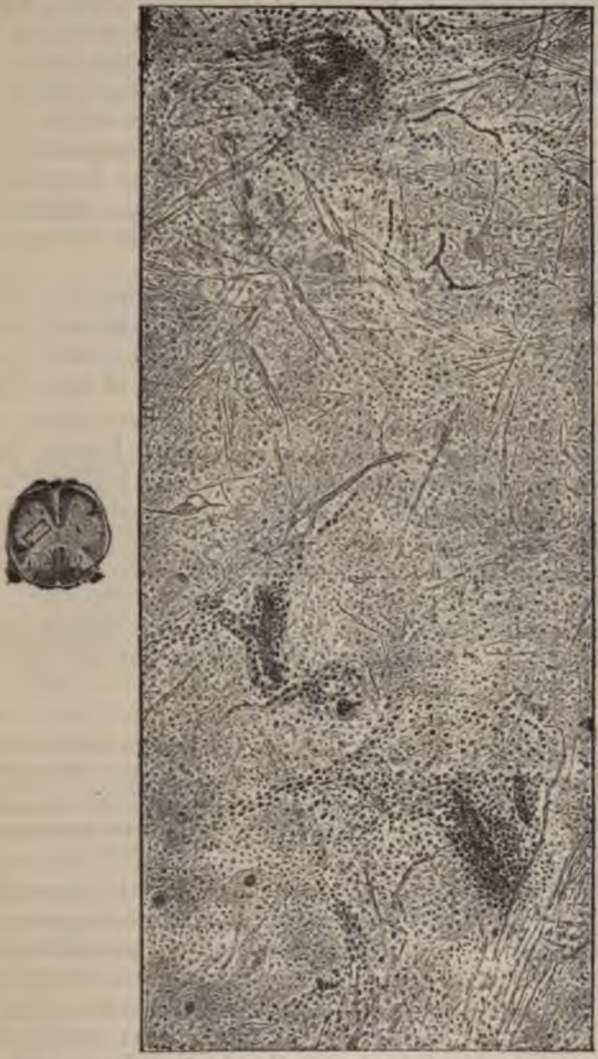

FIG. 117.—Acute anterior polio-myelitis in a child 14 months old. Dr. F. E. Batten's case. The affection commenced in the legs, but there were electrical changes and paralysis in the arms as well. The child died suddenly on the fourteenth day of the illness. The section made and lent by Dr. Batten shows marked engorgement of vessels, with occasionally actual extravasation, small-celled infiltration, and changes in the anterior horn cells.

inflammation may be seen. The posterior columns are always unaffected. In very severe cases there may be signs of slight local meningitis corresponding in position to the region chiefly affected. This region corresponds to the origin of the nerves supplying the muscles in which there is most wasting, and the anterior nerve-roots which arise from this part present the signs of acute degeneration.

Such changes indicate a general or interstitial inflammation of the grey matter. But some observations have revealed only alterations in the motor nerve-cells, which have presented granular degeneration, the matrix between them being quite unaffected.* This suggests that there may be a form of polio-myelitis in which the changes are essentially parenchymatous and not interstitial.

At a later period, years after the onset, the appearances presented accord with those described as commonly found in the early stage, allowance being made for the difference in time. The anterior cornu at one or more places is shrunken (Fig. 118), and in part stains deeply with carmine, in consequence of its nervous structures being replaced by connective tissue, in part less deeply in consequence of "granular disintegration" of the substance. The motor nerve-cells are absent, partially or entirely. Sometimes a few shrivelled cells remain, sometimes one or more groups may be unaffected; or one or two nerve-cells of normal appearance may alone remain (see Fig. 118). Small foci of disease may be seen in the unaffected part of the grey matter, and in these the motor cells may be smaller than normal; the intervening plexus may have perished. When one horn is much damaged, the other may merely present fewer nerve-cells than

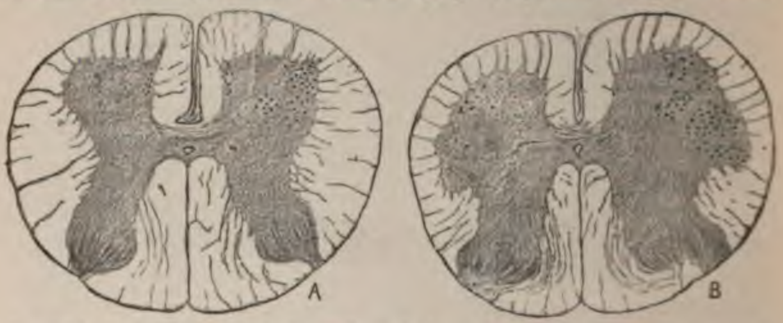

FIG. 118.—Atrophic spinal (infantile) paralysis. A. First lumbar; the left anterior cornu is smaller than the right, and its ganglion-cells have disappeared with the exception of the inner group. B. Fourth lumbar; the whole left half of the cord is smaller than the other, in consequence of the diminution in size of the ant. horn. Of the ganglion-cells only a few remain, belonging to the antero-lateral group. Similar changes existed throughout the lumbar enlargement.†

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* Rissler, 'Nord. Med. Ark.,' xx.

† For these sections I am indebted to Dr. H. Humphreys, of St. Leonards. The paralysis came on at one and a half years of age, two years before death, and involved most of the muscles of the left leg, those of the calf in greatest degree, so that talipes calcaneus had developed.

normal, with perhaps a little condensation of the interstitial tissue. The antero-lateral column is usually lessened in size, and may present slight sclerotic changes, especially in a zone contiguous to the grey substance, whence thickened septa may radiate into the white column. This is apparently due to the extension of an intense inflammation of the grey substance to the adjacent part of the white column. Sometimes a slighter sclerosis can be traced through the lateral column; this is seen chiefly in cases of long duration, and is probably secondary. With extreme damage to both cornua there may be ultimately great wasting of the white columns on each side.* In consequence of these alterations, the affected half of the cord is conspicuously smaller than the other, even to the naked eye, and the difference is greatest where the damage to the grey matter is most intense. The anterior nerve-roots at the most affected part are small and grey, and the degeneration of the motor fibres may be traced down the nerve-trunks. Often a few fibres present a normal appearance although the rest have perished; possibly from the sympathetic system.

The muscular fibres, in early cases, have been found narrower than normal, and in a state of granular degeneration, with an increase in the nuclei of the sheath and of the interstitial tissue. Granules and pigment masses accumulate between the sarcolemma sheaths. In extreme cases (those in which all electrical excitability is early lost) the degeneration proceeds to complete disappearance of the fibres, the place of which is occupied by fibrous tracts, developed partly from the sarcolemma sheaths and partly from the interstitial connective tissue. It is common to find, here and there, a fibre presenting normal appearances. Sometimes fat accumulates in the interstitial tissue, so that the reduction in the bulk of the muscle may be less than that of the fibres. In slighter cases, in which partial recovery takes place, some of the fibres probably regain their normal appearance, even though they have undergone slight granular degeneration. Usually, however, many fibres perish; those which recover are smaller than normal, and the original volume of the muscle is rarely regained. Although there may be a permanent increase in the interstitial tissue, this undergoes contraction and comes to occupy but a small bulk. Muscular fibres are often seen that are larger than normal, and this is not (as has been thought) a transient condition, for it has been observed seventy and forty-five years after the onset.† It is perhaps sometimes a true hypertrophy.‡

The bones have been found not only shorter than normal, but also smaller, with their angles rounded, the compact substance more uniform, and the Haversian canals smaller (Joffroy and Achard).

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* See 'British Med. Journal,' 1887, p. 187.

† Joffroy and Achard, 'Arch. de Méd. exp.,' 1889; Dejerine and Huet, 'Arch. de Phys.,' 1888.

‡ Dejerine. It is said to have been met with in muscles totally paralysed; but it is doubtful whether "total palsy" of a muscle excludes voluntary contraction of some fibres in it.

The brain has been almost always found normal. In one case with extensive paralysis, of long duration, some atrophy was found in the motor region of the cortex (Sanders). It is probable that some acute changes would be found in cases that die with the cerebral symptoms already described.

No observations have as yet been made on the acute primary polyneuritis that seems occasionally to coincide with the inflammation of the cord, perhaps sometimes to exist alone without any cord affection.[*] It is probable that, in the cases in which the nerves are tender in the middle of their course, the sheath will be found inflamed, and only the motor fibres will present such signs of secondary degeneration as always result from the lesion of the cord.

Pathology.—Both the character of the lesion and its mode of onset suggest that it is inflammatory in the majority of cases. In rare instances, as already stated, the almost instantaneous onset makes it probable that the lesion is different in character, is vascular in its nature, probably hæmorrhage. It is likely that these cases are really hæmorrhagic myelitis, in which the usual initial vascular disturbance is so severe as to produce an extravasation. Signs of inflammation do not precede the sudden onset.

The symptoms are well explained by the pathological facts that have been ascertained, extended by the simple inferences that are indicated by the symptoms. The lesion interrupts the motor path, causing immediate palsy, which results from damage to the cells and nerve-plexus of the grey matter, whether slight or severe. The recovery of the slightly damaged structures restores the voluntary path, and even if the fibres from the cells degenerate, they seem to regain normal nutrition by regeneration when the cells recover. But when the motor cells are destroyed the fibres from these degenerate permanently. As the slighter damage is usually far wider than the destruction, the initial palsy is more extensive than the persistent atrophy, and the extent of such is purely a matter of this relative area of trifling and severe degree of lesion. Nerve-cells that are destroyed are never renewed, and hence the permanence of the muscular wasting that depends on such destruction. When a few nerve-cells escape destruction, a few nerve-fibres recover and a little muscle-tissue remains, too slight, it may be, to be of use. The occasional interference with the conducting tracts in the white columns is explained by the obvious extension of the inflammation, which, when very intense, may even spread through all the structures of the cord, as in the rare cases in which sensation, as well as motion, is lost. But such cases, which are most rare, differ from true transverse myelitis in the rapidity with which the damage restricts itself to its special seat, and sensory conduction is restored. The fact that the disease of the sensory tracts is only secondary and trifling, is emphasised by the fact that even in these cases there are never the acute bedsores that would surely ensue in a case of true transverse or total myelitis of corresponding seat.

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[*] As was probably the case in two cases of paralysis in children shown to the veian Society of London by Dr. Leonard Guthrie, 'Lancet,' 1897, vol. i, p. 448.

The observations mentioned on p. 412 suggest that there are two varieties of the inflammation of the grey matter, such as we have recognised in myelitis generally; a parenchymatous inflammation consisting in a primary change in the nerve-cells, and an interstitial or general inflammation, as in the common forms of myelitis, involving the nerve-cells only as one of many structures equally damaged by a process that has no special relation to the nerve-elements. The distinction is important, because we should expect to find, in the former cases, a wider slight initial affection than in the others, and finally a more definite relation to function in the parts diseased.

The true pathology of the malady, its relation to its causes, is still obscure. The one salient fact that we can discern, though scarcely define, is the evidence of a blood-state afforded by the general symptoms. That these symptoms are not the consequence of the local inflammation is shown by their disparity in degree, and difference in time. It is probable, therefore, that the spinal lesion is not the cause of the constitutional disturbance, but is rather an effect of the cause of the latter. It seems impossible otherwise to understand the extreme variation in the two forms of disturbance. But we have no evidence as to the nature of the blood-state, and there are no other effects, commonly associated with the myelitis, to indicate the general pathological tendency of its cause. The closest analogy is with some forms of neuritis, and this, as we have seen, may co-exist with the spinal lesion, as a consequence, at any rate, of exposure to cold. Indeed, it is probable that the inflammation of the nerves is the chief or even the only morbid process in some of these cases. On the other hand, it must be noted that in the epidemics of the disease, there were, besides cases of spinal cord disease, simultaneous cases in the same places in which the evidence pointed to an analogous inflammation in the cerebrum. In one epidemic, the American one, the lower animals also suffered (see p. 416). The variations, alike in the general symptoms and in the inflammation of the cord, and especially the indications that the latter may be either parenchymatous or interstitial, suggest that the causal blood-state also varies in different cases. We have no indication of the way in which the remarkable relation to season is produced, whether by predisposing the individual or favouring the development of some toxic agent. We must, however, recognise as an essential element the predisposing influence of age, which we may associate with the facts that the structural development of the nervous system is complete, but the function of the nerve-elements must lack the stability that comes only from continued use, and that the period is generally that which coincides with the first serious demand on the [illegible] of the cord. We may remember [illegible] all function entails, and the readiness [illegible] of children is disturbed. Lastly [illegible] in more than one member of the same [illegible] disposition of the system to react in a [illegible] agencies, while a special feature in [illegible] the occasional epidemic character of a [illegible] for ascribing to it an "infective" [illegible] available suggests that it is probably due to [illegible] blood analogous to that which seems to [illegible] probably distinct from it—a change [illegible] by the effect of heat, the result of some [illegible] which we cannot yet understand, and [illegible] these effects some organismal cause. This [illegible] the action of traumatic influences in [illegible] Such a glimpse of its pathological relations [illegible] obtain.

[illegible] presents any difficulty except in the early [illegible] paralysis is passing away, and the wasting is [illegible] case is sufficiently evident, and is cor- [illegible] reactions, by the loss of reflex action, and [illegible] impairment of sensibility. At the onset, [illegible] may readily be misinterpreted. The most [illegible] too much importance to vomiting, and [illegible] simply gastric. General disturbance is [illegible] general cause until nervous symptoms [illegible] then there is risk of error. On the one [illegible] diagnosis of a general disease is not always [illegible] appears, but the latter is regarded as [illegible] hand, especially in young children, the [illegible] overlooked at first, and it is supposed [illegible] because it is prostrate. This error will [illegible] that mere prostration seldom causes [illegible] *a fortiori* it does not produce local immobility. [illegible] and the loss of power persists and increases, [illegible] is always unmistakeable. The initial general [illegible] be regarded as part of the disease unless there [illegible] its independent nature.

[illegible] of mistaking paralysis for prostration is con- [illegible] other hand, the general symptoms are as [illegible] as in the case of children. The rheuma- [illegible] common, are usually regarded as evidence of [illegible] especially when the affection follows exposure to [illegible] pains are not localised in the joints

[illegible] 1896, p. 1120; Buccelli, 'Neur. Cent.,' 1897, p. 798; [illegible] Assoc.,' January, 1896; Pasteur, 'Trans. Clin. Soc.,' and especially when they are spontaneous, and not influenced by movement, the possibility of their spinal (or nerve) origin should be remembered; and other indications of spinal mischief, such as local loss of power, tingling or formication, should be carefully watched for and receive due weight.

Of other spinal cord diseases, all chronic lesions are distinguished at once by the onset. From other acute diseases, moreover, the distinction is only difficult in the early stage. As soon as the initial palsy begins to lessen, and the muscles in one part lose faradic irritability and begin to waste, the nature of the case admits of no doubt. The diagnosis may be made with certainty after the end of the first week by the electrical reaction. If we find loss of faradic irritability, it is certain that the characteristic alteration in voltaic irritability will follow. The isolated induced shocks should be used instead of the serial "current," because any harmful stimulation of the sensory nerves is thereby avoided. (See Diseases of the Nerves.)

Acute transverse myelitis is only simulated when the inflammation of the grey matter is bilateral and so intense as to extend to the white columns, but the age of the patient generally suggests correctly the nature of the disease. The myelitis of childhood is polio-myelitis. The pseudo-transverse variety—polio-myelitis, transverse through its intensity—is seated in one of the enlargements, usually the lumbar; the true transverse form usually occurs in the dorsal region. Real difficulty is confined to adults, in whom there is extensive cervical or lumbar myelitis involving the grey matter. In young adults, polio-myelitis is more likely; this is indicated also by what is best described as a regressive onset in contrast to one that is progressive. In the former a wide extent is quickly reached, and the symptoms then tend to lessen; in the latter the morbid process is a longer time reaching its height: in the former the symptoms clearly show that the most severe affection is of the anterior grey matter; in the latter, severe anæsthesia or trophic disturbance usually points to an equally intense lesion of the other parts of the cord.

The separation of subacute from acute myelitis of the grey substance is, to a large extent, arbitrary. The symptoms are similar, but the onset is less rapid and occupies more than a week. The distinction of multiple neuritis is from the subacute rather than the acute spinal atrophies, and is considered elsewhere.

The diagnosis from paralysis of cerebral origin is usually easy. In cerebral palsy there is never loss of faradic irritability or extreme muscular wasting, or loss of the muscle-reflex action. In the spinal affection there is no trace of the mobile spasm that is common after infantile hemiplegia. Any cerebral symptoms which may attend the onset of polio-myelitis are subordinate in significance to the state of the muscles as evidence of the permanent lesion. Convulsions at the onset of infantile spinal paralysis are general; those that result from a cerebral lesion are usually unilateral or commence locally. The chief difficulty arises when arm and leg are involved on the same side. These cases are rare, and present no difficulty if attention is paid to the nutrition and reaction of the muscles.

The exceedingly slow onset of pseudo-hypertrophic paralysis, developing gradually, as it does, with the child's growth, ought to render its confusion with polio-myelitis impossible. A reasonable doubt can only arise in a slight case in which the onset was unnoticed or forgotten. I have once known slight atrophic palsy of the extensors of the knee to induce the habit of putting the hand on the knee in rising from the ground—an action often thought to be peculiar to the pseudo hypertrophic disease, but acquired when weakness of the extensors occurs in early childhood from any cause.

Diseases outside the nervous system which have been mistaken for infantile paralysis are chiefly those in which local pain interferes with the movement of the limbs, and the child is young. I have known, for instance, the mistake to be made in the case of hip-joint disease, necrosis of the femur, and the affection termed "scorbutic rickets," in which there is enlargement of the shafts of the long bones, extreme pain on movement, and spongy gums. In all these cases a careful examination will show that movement is interfered with only by the pain; there is no actual paralysis, and there is no interference with reflex action. The preservation of the knee-jerk is often of great significance; it at once excludes atrophic palsy in any case in which the extensors of the knee seem feeble.

The points above described should always enable the diagnosis to be made. It is impracticable to enumerate the distinctions from every disease with which confusion is possible, for experience shows that there is no form of palsy with which a common disease, such as this, is not at some time confounded.

PROGNOSIS.—The danger to life is probably greatest when there is severe constitutional disturbance, which may even precede the development of the characteristic paralytic symptoms. In the stage of paralysis there is peril only when the chief disease is in the cervical region, and respiration is interfered with; but this danger is small unless an attack of bronchial catarrh is intensified by the palsy. Definite cerebral symptoms involve danger proportioned to their character and degree, but it is generally less in reality than in appearance. In the vast majority of cases the disease involves no immediate danger to life. But children are left with little power of resistance to other morbid influences, and occasionally succumb to some other illness, as an acute specific disease, or an attack of bronchitis, a few weeks or months after the onset of the paralysis.

As soon as the paralysis has become stationary, *i. e.* has not increased for twenty-four hours, the danger of further extension is small. But the question at once arises, and is anxiously asked,—What will be the permanent condition? Will there be lasting paralysis? An answer cannot be given until the end of the first week or ten days, and then only by means of an electrical examination. Whatever muscles, at the end of that time, have lost faradic irritability will certainly waste, will remain for a long time paralysed, and will probably be permanently affected in some degree, slight or severe. On the other hand, if there is no loss of irritability at the end of ten days, but it is apparent at the end of a fortnight or three weeks, the wasting will be slighter in degree, and some ultimate recovery may be anticipated even in the most affected part. Where there is no loss of irritability the paralysis will pass away in the course of a few weeks, or at most of a few months. When faradic irritability is lost early and completely, the wasting will be rapid and great, and it is unlikely that there will be much recovery. The return of faradic irritability that has been lost is a favorable indication; it signifies nerve-regeneration, and will be followed by an increase in voluntary power. Without an electrical examination it is necessary to wait longer before a prognosis can be given, until distinct wasting on the one hand, or improvement on the other, indicates the regions in which the paralysis will persist and in which it will pass away. Even then the prognosis cannot be so definite.

In the chronic stage the prospect of ultimate recovery depends on the rate at which wasting developed, on the electrical reaction, and on the duration of the case. Where there is no sign of returning power at the end of three months very little recovery will occur; the nerve-cells are destroyed, their renewal is impossible. The preservation of voltaic irritability (that of the muscular fibres) is so far satisfactory that it shows there has been no destructive degeneration of the muscles; and if voluntary power is increasing, it indicates favorable conditions for its exertion, but it does not lessen the grave significance of persistent palsy and the absence of faradic irritability, which indicate persistent nerve-degeneration. On the other hand, if, at the end of one or two months, some faradic irritability can be still detected, although low in degree (*i. e.* elicited only by a strong current), improvement is probable, and may become considerable. It is necessary, in the case of children, to remember, and it is important to warn the friends, that the growth of the most affected limb will be hindered, and that this, in the case of the leg, may render the effect of the paralysis more obtrusive by its interference with the gait. Otherwise they are distressed by what seems to them an increase in the disease, but is really compatible with continued improvement.

Treatment.—The treatment of the acute stage of the disease is essentially the same as that of myelitis, already described, and the rules and principles already stated need not be here repeated. In the initial stage it should be that of the general state, guided by any special causal indications that may be detected; such as free sweating, followed by salicylate of soda or salicin, in a case distinctly due to exposure to cold. We may hope that future observations will afford us some indications regarding the means of counteracting other blood-states that apparently exist at the onset, and are concerned in the production of the lesion. At present we are without this help, and can only treat the initial stage as we should any other local inflammation, directing our treatment to the symptoms that may be present, and the conditions that apparently underlie them. The child should be kept at perfect rest, on the side, so that the spine is not the most depending part. Warmth may be applied over the affected part of the cord by poultices or fomentations. The marked relief these give, when there is spinal pain, make it probable that they exert a beneficial influence in all cases.

In such a disease—in which there is a natural tendency for the morbid process to cease to spread, and then to lessen in extent—the difficulty of ascertaining the effect of treatment on the lesion of the spinal cord is very great. Full doses of belladonna or ergot have been credited with the control and arrest of the morbid process, but only on the evidence of a coincidence in isolated cases, the value of which is small in a disease so irregular in its course. Either may apparently be employed, however, without fear of harm. But probably the measures mentioned above directed to the local inflammation have far more influence than drugs on the morbid process. As in all acute diseases of the nervous system, the chief room for effective treatment rests with the practitioners who see the cases in the early stage.

When the acute onset is over and the spinal lesion has become stationary the careful management of the early stage should be continued for a time, because a relapse or recurrence, however rare, is not entirely unknown. Such care is especially needed in cases in which the constitutional disturbance has been prolonged, or has continued after the onset of the spinal symptoms, or in which the latter have come on in distinct stages. Under these circumstances, perfect rest should be maintained for one or two weeks more. The same prolonged care is needed when there is persistent tenderness in the limbs, or any indications of an independent neuritis. Where wasting is taking place, some slight tenderness of the nerves and of the muscles must be expected to accompany the process, and this, being purely secondary in nature, does not call for special treatment.

When the acute stage is over, and the condition of the patient is stationary or improving, tonics are generally both useful and needed, especially iron and quinine. Strychnia may be given in all cases, but it should be commenced only three or four weeks after the disease has become stationary. Although clear proof of its utility is not forthcoming, and cannot indeed be expected in a disease in which merely damaged nerve-elements always tend to recover, the drug has a definite influence on the nutrition of the structures that are specially affected, and it is reasonable to suppose that it promotes their recovery, and is capable of rendering this greater. But an agent that has so powerful an influence on function (and therefore on nutrition) is likely to do harm, rather than good, until normal conditions are being restored. If function cannot respond properly to a stimulus, it is not likely that nutrition can be influenced aright. Hence, the more severe the lesion the longer time should elapse before strychnia is commenced, and the smaller the dose that should at first be given. It is probably never either necessary or desirable to give it by hypodermic injection in this disease.

The disease frequently occurs at the age at which any illness causes the hindrance to general development that constitutes the condition known as rickets, and this fact should be remembered in treatment. In the case of children who are still in the period of the first dentition, it is, therefore, wise to anticipate the danger by giving, during convalescence, those agents that have most influence in counteracting the tendency, especially cod-liver oil and iron; the lacto-phosphate of lime and iron is especially useful.

The use of electricity is an important part of the treatment, although for reasons very different from those that first led to its use, and it is desirable that they should be clearly comprehended to secure its benefit and prevent its harmful or useless employment. There is no evidence that it can or does influence the process of recovery of the damaged elements in either the spinal cord or the nerves. Moreover, as long as the nutrition of these is actively disordered, its stimulating influence is likely to increase the derangement rather than promote the restoration of a normal state. The reasons for its use depend on the fact that the disease entails nerve-degeneration, and are essentially the same as those that justify and determine its employment in other nerve lesions, and have been already explained. The muscular fibres whose nerves are degenerated suffer changes in nutrition, and ultimately perish if no nerve-regeneration occurs. While the influence normally exerted by and through the nerve-fibres is in abeyance, the muscles are destitute of this influence, and without any functional stimulation. If they are excited from time to time by electricity, their sensitiveness to stimulation is distinctly increased, and this not only to electricity, but to the voluntary stimulus. This is clearly shown by cases that have been untreated; the muscles may not respond at all to the first application, but when they have been galvanised two or three times a distinct contraction may be obtained, and, within a week, some voluntary power may return. Wherever cell and fibre have perished, nothing that electricity effects can be of service; but where there has been damage, not destruction, and the nerve-fibres slowly recover, but regain their influence on nutrition, and their capacity for conveying impulses, only after some months, the failure of muscular nutrition may be disproportionately great, and may even render useless some regained nerve-power. This result electricity is probably able to prevent. It does not, as far as we can perceive, prevent or even lessen the visible wasting of the muscles; it is powerless to counteract the effects of a destructive lesion; but it does seem to hasten recovery from the effects of partial functional energy of the grey matter of the cord. We may remember also the vascular activity that all function entails, and the readiness with which the vaso-motor system of children is disturbed. Lastly the occurrence of the disease in more than one member of the same family indicates a congenital disposition of the system to react in a similar manner to certain external agencies, while a special feature in the latter may perhaps explain the occasional epidemic character of a disease, and furnish grounds for ascribing to it an "infective" character.* The evidence available suggests that it is probably due to some chemical change in the blood analogous to that which seems to cause rheumatic fever, though probably distinct from it—a change excited by cold, disposed to by the effect of heat, the result of some derangement of metabolism which we cannot yet understand, and perhaps having underlying these effects some organismal cause. This hypothesis does not exclude the action of traumatic influences in determining the disease. Such a glimpse of its pathological relations is all that we can at present obtain.

DIAGNOSIS.—This rarely presents any difficulty except in the early stage. When the initial paralysis is passing away, and the wasting is distinct, the nature of the case is sufficiently evident, and is corroborated by the electrical reactions, by the loss of reflex action, and by the absence of any impairment of sensibility. At the onset, however, the symptoms may readily be misinterpreted. The most common error is to attach too much importance to vomiting, and to regard the attack as simply gastric. General disturbance is naturally ascribed to some general cause until nervous symptoms manifest themselves. Even then there is risk of error. On the one hand, as we have seen, a diagnosis of a general disease is not always relinquished when paralysis appears, but the latter is regarded as secondary. On the other hand, especially in young children, the existence of paralysis is often overlooked at first, and it is supposed that the child does not move because it is prostrate. This error will not be made if it is remembered that mere prostration seldom causes total immobility, and *à fortiori* it does not produce local immobility. When the pyrexia ceases, and the loss of power persists and increases, the existence of paralysis is always unmistakeable. The initial general disturbance must then be regarded as part of the disease unless there is the clearest evidence of its independent nature.

In adults, the danger of mistaking paralysis for prostration is considerably less, but, on the other hand, the general symptoms are as likely to be misinterpreted as in the case of children. The rheumatoid pains, which are so common, are usually regarded as evidence of acute rheumatism, especially when the affection follows exposure to cold. Whenever rheumatoid pains are not localised in the joints and especially when they are spontaneous, and not influenced by movement, the possibility of their spinal (or nerve) origin should be remembered; and other indications of spinal mischief, such as local loss of power, tingling or formication, should be carefully watched for and receive due weight.

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* See Medin, 'Neur. Cent.,' 1896, p. 1120; Buccelli, 'Neur. Cent.,' 1897, p. 798; Sowerby, 'Journ. Am. Med. Assoc.,' January, 1896; Pasteur, 'Trans. Clin. Soc.,' 1897.

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The diagnosis from paralysis of cerebral origin is usually easy. In cerebral palsy there is never loss of faradic irritability or extreme muscular wasting, or loss of the muscle-reflex action. In the spinal affection there is no trace of the mobile spasm that is common after infantile hemiplegia. Any cerebral symptoms which may attend the onset of polio-myelitis are subordinate in significance to the state of the muscles as evidence of the permanent lesion. Convulsions at the onset of infantile spinal paralysis are general; those that result from a cerebral lesion are usually unilateral or commence locally. The chief the calf muscles, often aggravated in consequence of the owth of the limb. Something may be done during the ubbing for the prevention of deformities. While the con- scles are rubbed upwards they should be extended gently Thus, if the calf muscles are shortened, the foot should be ssed upwards while the calf is rubbed. A slipper with s to a knee-band may be worn at night, often with great e developed deformities, due to great shortening of the surgical treatment by splints or tenotomy is usually necessary, often important because it enables the patient to use the limb in ways that would be impossible without it.

For this and other reasons mechanical appliances are of unquestionable value. Counteracting deformities and supplementing weak muscles, they often enable a child to walk who could not do so without their aid, and power is often increased to a remarkable extent in indirect ways. In all these cases, however, the instruments require frequent attention and alteration to adapt them to the changes due to growth, and patience is required in the management of these cases through their long and tedious course.

Two facts deserve a final emphasis. At the end of six months all possible recovery is nearly completed, and certainly at the end of a year the lesion has become a cicatrix, and further improvement will be merely by the slow growth of the muscle that has recovered, under the stimulus of use. Secondly, this process of slow improvement as the result of use will go on for years,—will go on whatever treatment is adopted, and whether special measures are employed or not. The therapeutic specialist who carries on his treatment month after month will claim it as his achievement, but it would occur equally without his aid. From its nature, however, as already stated, it may probably be always augmented by some contrivance to increase the influences that are really improving the power of the muscles.

Subacute and Chronic Atrophic Spinal Paralysis

(SUBACUTE AND CHRONIC POLIO-MYELITIS).

Under this designation cases have been described of a miscellaneous character, in which paralysis, followed by muscular atrophy, comes on less rapidly than in the acute form, its development occupying from one to thirty days in the cases which are termed "subacute," and from one to six months or even more in those which are called "chronic." Many cases included under this name differ much in their characters, and are sometimes described as "chronic myelitis;" but among the cases placed, even until lately, in this group, are forms of multiple neuritis. Most subacute atrophic palsies are due to nerve lesions, almost all, indeed, which present symmetrical palsy.

The cases of spinal atrophic paralysis, in which the onset is not sudden, may be placed in four classes.

(1) Cases of subacute polio-myelitis which differ from the acute form, already described, only in their less rapid development. They present the same initial general disturbance and wide distribution, and the same recovery, except in a limited region in which muscular wasting occurs. They are thus distinguished from polyneuritis, and also by the irregular distribution of the symptoms. Most of the sufferers have been adults. Little is known of the exciting cause of this form further than that it certainly sometimes follows exposure to cold, and is probably sometimes the result of a toxæmic influence. The symptoms and general history of these cases present no important difference from the acute form.

(2) Cases are met with that differ from the type just described in the fact that their course is progressive. The onset is subacute or subchronic, occupying from a fortnight to several months, but instead of arrest followed by improvement, the more or less rapid onset is followed by slower gradual increase, until at last wide-spread chronic muscular atrophy is developed. Many of these are really cases of progressive muscular atrophy with a subacute onset. For example, I have more than once known a case of this disease, ultimately typical, to begin by a subacute atrophic paralysis of the extensors of the wrists. These cases will be considered in the account of that disease. Others present differences from that malady and symptoms of more irregular character, which show that the lesions are distributed through various structures in the cord, and these must be regarded as cases of chronic myelitis involving the anterior cornua. This form also is chiefly confined to adults. It occasionally results from cold, and sometimes from injury, as a fall on the back. Intemperance and venereal excesses are also said to cause it. The wide-spread muscular atrophy which sometimes results from lead-poisoning is probably of this nature. The muscles at first present the degenerative reaction, or normal faradic irritability with increase to voltaism; sometimes, however, there is a loss to both faradism and voltaism. The sphincters usually escape. In many cases the affection exhibits a persistently progressive character; muscular atrophy slowly increases and extends, until the case ultimately resembles one of progressive muscular atrophy, and the patient dies at the end of one or two years from exhaustion, or from interference with the respiratory movements. In other cases the atrophy, after slowly progressing for many months, becomes stationary, and considerable improvement may ultimately take place. This course is seen especially in traumatic cases. Few observations on the pathological anatomy of the affection have been made. Cornil and Lépine found, in one case, at the end of four years, softening of the lower part of the spinal cord, chiefly in the anterior cornua, disappearance of the ganglion-cells, increase of the connective tissue, and sclerosis of the white substance around the anterior horns.

The diagnosis from the first form rests on the slow onset and pro-

ACUTE ASCENDING PARALYSIS.

Ascending paralysis, which commences in the legs and ascends to the muscles of the trunk, the arms, muscles of the neck, the diaphragm and the pharynx, is a feature of several diseases—meningeal hæmorrhage, ascending myelitis, &c.; but it occurs also in cases in which the spinal cord, after death, appears healthy to both naked-eye and microscopical examination. Such cases have, however, certain characteristic features which make it desirable to distinguish the condition from other diseases, and the affection has, therefore, been provisionally termed "*acute ascending paralysis*," in the absence of any evidence of its nature. It was first described by Landry in 1859, and hence is often called *Landry's paralysis*. Acute ascending paralysis is a most formidable malady, most cases proving fatal in a few days. Its nature is mysterious, but recent discoveries regarding multiple neuritis have disclosed instructive analogies between the two affections. These have, indeed, led some observers to the opinion that this ascending paralysis, without organic central lesion, is an affection of the nerves.[*] As we shall see, it is not probable that this opinion is correct, although it is possible that the nerves, as well as the centres, are sometimes affected.

Causes.—The etiology of the disease resembles, in general, that of acute multiple neuritis more nearly than that of any other affection. The disease affects males more frequently than females. It occurs chiefly between twenty and forty years of age, but has been observed in rare cases in older and younger persons, and even in children. Some sufferers have been the subjects of alcoholism. Severe exposure to cold has been the apparent cause in many instances. In other cases the disease has occurred under conditions such as cause toxæmic states, or after the occurrence of some known blood disease. Thus it has occurred during convalescence from some general disease, smallpox, diphtheria, typhoid fever. It has also followed influenza and febrile diseases of obscure nature. In these cases, it should be noted, the disease has followed at an interval of a week or a few weeks. It also sometimes succeeds some traumatic process, such as a wound, and here also after an interval, and usually when the wound was apparently healed. I have known it thus to follow an attack of pelvic cellulitis. In these respects it closely resembles polyneuritis; and it has been observed, in severe form, in the subjects of old alcoholic neuritis. The disease has been repeatedly observed in the subjects of syphilis, and has apparently been arrested by the treatment for this disease.[†] The circumstance that when some malady or wound pre-

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[*] See Ross and Judson Bury, 'Treatise on Peripheral Neuritis.'

[†] Much weight should not be placed on this fact as proof of causal relationship, because (apart from the fallacy of independent cessation) it is probable that the treatment that is effective against the virus of syphilis is also effective against other blood-states that depend on agents of similar nature.

and not until all paralytic symptoms have disappeared has the knee-jerk returned.

In spite of the early flaccidity of the muscles, if life is prolonged they rarely present either wasting or change in electrical irritability. A trifling reduction in size may occur, but there is no muscular atrophy such as occurs in polio-myelitis, and even after several weeks the most careful examination fails to reveal any abnormal electrical reaction—a very important feature of the disease. Exceptional cases, which do present the degenerative reaction in the muscles, seem more closely allied to a universal polio-myelitis, from which, indeed, this disease cannot be sharply separated. The sphincters, moreover, escape in the vast majority of cases, but not in all. There is no tendency to the occurrence of bedsores. The cerebral functions are not usually involved, and the state of the patient who, with unimpaired intellect, cannot express himself either by speech or gesture, is painful in the extreme. In exceptional cases, however, there is mental dulness or slight delirium, as if from the influence of a blood-state on the brain. In girls this may take the form of hysterical manifestations, by which the nature of the disease may be masked.

As a rule there is no elevation of temperature, even during the rapid development of the symptoms, but in one or two cases, which were not fatal, brief pyrexia attended the onset, and in a few others moderate febrile disturbance (2° or 3°) occurred at a late period. Profuse sweating has been occasionally noted. Enlargement of the spleen (first noted by Westphal), such as occurs in other acute diseases, is very frequent.

Thus the chief feature of the disease is an almost purely motor paralysis, progressive in character, with relaxation of the muscles during the acute stage of the disease. It usually has an ascending course, but irregular cases are met with in which the order of invasion is varied. Thus the arms are occasionally involved before the legs, the diaphragm before the intercostals; the bulbar symptoms have been known to precede the others, and I have seen them reach a high degree with a fatal involvement of the cardiac centre before the upper muscles of respiration were affected, or the hands quite powerless. Analogous cases have been recorded by others, the special features and course being those characteristic of the disease, In some cases, however, of irregular course, changes have been found in the electric irritability of the muscles; it is probable that, in such cases, the peripheral nerves are affected, although we cannot, on this account, at present separate these from the typical cases of the disorder—a point that will be explained in connection with its pathology.

The disease varies considerably in the rate of its progress. It may run its course and end fatally in forty-eight hours. Death usually results from either respiratory or cardiac paralysis—the latter in the cases of irregular course. A large proportion of the fatal cases last less than a week. On the other hand, the disease may only attain its

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and Marie, Bailey and Ewing.
Wochenschr.,' 1890, No. 38.

similar course. It has, indeed, been conjectured that the nerves are always the structures impaired, but this exclusive view is disproved by the normal state of the nerves in cases in which the grey substance of the spinal cord has been found to be changed and by the common absence of altered excitability of the muscles, since the motor nerves seem never to escape invariably in peripheral neuritis of any variety, however common their freedom from affection in the type. But the nerves suffer also in some cases, and there seem to be gradations to the cases of acute multiple neuritis of ascending course described at p. 162. All the facts point to the conclusion that the disease is due to a toxin which has a specific action on the motor neurons in the spinal cord, chiefly on the anterior grey matter, sometimes also affecting the motor nerve-fibres proceeding from them. Sometimes it has a wider influence on the spinal cord, and its effects can be traced into the white substance or the posterior horns, possibly as an extension of the inflammatory changes it induced. These are similar to those that attend any acute disturbance of function; the toxic influence is mysterious in nature and origin, but seems to resemble that which causes polio-myelitis, in which the morbid process presents variations and may also involve the nerves. The toxine may be produced by the agency of bacteria, but the evidence of this is inconclusive; some cases have followed exposure to cold.

Diagnosis.—The disease is recognised by the rapid development of paralysis, usually but not invariably ascending, with relaxation of the muscles, and with loss of reflex action, without considerable pain or, generally, any loss of sensation, and if the patient survives, without wasting of the muscles or change in electrical irritability. The latter characteristic distinguishes the disease from acute atrophic paralysis, while the absence of pain in the back and of spasm is a distinction from meningeal hæmorrhage. The diagnosis from general ascending myelitis has been already considered; it rests especially on the involvement of all the functions of the cord in inflammation. The distinction from multiple neuritis has been mentioned in the account of that disease (p. 174), but, it may be, is not always absolute, since the nerves probably suffer, as well as the cord, in some cases of ascending paralysis. Further, the cases of multiple neuritis simulating this affection, the nature of which has been demonstrated pathologically, have usually run a more prolonged course, several weeks or even months. The distinction from polyneuritis, founded on the course of the ascending form, is subject, however, to the reservation that while ascension to the arm through the trunk is a feature of the central disease, this is not excluded by a more irregular course, and the character of the individual symptoms must be taken into consideration (see also p. 429).

Prognosis.—The affection is one of extreme gravity. The danger to life is in proportion to the interference with respiration and with the functions of the medulla oblongata, especially with the cardiac centre, and also to the rapidity with which the palsy comes on. But the cases which develop with comparative slowness are not devoid of danger. Even when the symptoms only reach their height at the end of three or four weeks, death may occur in the same way as in the more rapid cases. On the other hand, recovery has been known although the patient has lost all power of motion at the end of the second day. The danger is great as long as the symptoms are increasing, and only when distinct improvement can be recognised is it justifiable to anticipate recovery. The earlier the bulbar symptoms appear, the more serious is their significance. Mental symptoms also increase the gravity of the prognosis, as they usually indicate a severe blood-change, the effects of which on the nervous system are likely to reach a high degree.

TREATMENT.—During the early stage of an attack of acute ascending paralysis, in the absence of other indications, the treatment should be that suitable for myelitis, since, at the onset, the diagnosis between the two diseases can never be certain. A warm bath, or still better, a vapour bath, should be given if the symptoms followed exposure to cold. It should be followed by counter-irritation over the spine by a long, narrow mustard plaster. More energetic counter-irritation, even the actual cautery, has been recommended. The body should be kept in as perfect rest as possible, and on the side rather than on the back. In very few cases have drugs appeared to exert any influence on the course of the disease, and the malady is so rare that experience accumulates slowly. Salicylate of soda seems to deserve a trial in cases that follow exposure to cold. Ergotin has been given, and one case in which it was used deserves special mention. The patient was a man aged fifty-seven, who, a week after exposure to cold and wet, complained of a feeling of weight and weakness in the legs; the temperature rose to 103°; the loss of power gradually became complete in the legs and spread to the arms, without loss of sensation. At the end of the second day there was difficulty in swallowing, in articulation, and in breathing, and death seemed near. Ergotin was given every hour, and during the night the patient took twenty grains. In the morning the bulbar symptoms were better, the arms stronger, and there was a trace of motor power in the legs. The patient rapidly improved, and at the end of a week was well. If swallowing becomes difficult, care must be taken to administer a sufficient amount of nourishment, either by the rectum or by the nasal tube. A few cases have been recorded in persons who had had syphilis, in whom arrest of the disease followed the administration of iodide of mercury.

The chief lesson of modern research is certainly that we must look for the means of effective treatment to the neutralisation of the toxic influence on which the malady apparently depends, but at present nothing has been ascertained regarding antidotal agents available in the acutely active stage of these toxæmic states. It is, however, probable that mercury is capable of doing good, and that when the causation is obscure, and the disease develops slowly enough to permit mercurial treatment, it is wise to adopt this irrespective of the fact of preceding syphilis. In cases that follow traumatic lesions, especially if there have been any indications of septicæmia, full doses of perchloride of iron offer, I believe, the best means of destroying the activity of the blood-state. The general and local treatment should be the same as in acute myelitis.

PARALYSIS FROM LESSENED ATMOSPHERIC PRESSURE; DIVERS' PARALYSIS; CAISSON DISEASE.

Divers, and especially those who work in caissons, at such a depth beneath water that they are exposed to considerable pressure, may become paralysed soon after their return to the surface.* Apoplectic attacks and hemiplegia also sometimes occur, sometimes transient mental disturbance, or deafness, or bleeding from the nose, but paralysis of the legs is by far the most common effect, and it is, therefore, clear that the spinal cord suffers in greater degree than any other part of the nervous system. Miners have been said to suffer also, but this is doubtful, because a pressure equal to at least an additional atmosphere seems necessary for the production of the symptoms. Most of the subjects of the disease have worked at a depth of from forty to ninety feet below the surface of water, and under a pressure of two to four atmospheres. In the extensive works involved in laying the foundations of bridges, in which many men have been employed, a considerable proportion of the workers have suffered in some measure; but severe degrees of affection are rare, because the conditions under which they occur can be avoided. They only occur in those who have been exposed for more than a certain time, which is the shorter the greater the pressure. At a depth of ninety feet beneath the surface immunity from severe symptoms (and commonly from all symptoms) is obtained by reducing the periods of work to an hour. The danger is greater in those who have had several previous periods of work on the same day, and especially if slight symptoms have been experienced after one of these. It is also greater in those unaccustomed to the conditions. I have met with one case of hemiplegia in a diver after only half an hour's work in a well at ninety-six feet, but the man had been down twice before with only intervals of rest of half an hour, and the attack passed off in the course of an hour. In another case paraplegia occurred after working for an hour under only fifty feet of water, but the man had worked on the preceding day at a depth of one hundred and twenty feet below the surface. It is evident, therefore, that the power of resisting the dangerous influence varies, and that predisposition to suffer may be caused by the conditions which also induce an attack.

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* See, on this subject, Babington and Cuthbert, 'Dubl. Quart. Journ.,' 1863, p. 312 (cases at Londonderry); Eads, 'Med. Times and Gaz.,' 1871, p. 291 (cases at St. Louis); Leyden, 'Arch. f. Psychiatrie,' ix, Heft 2; and Moxon, 'Lancet,' 1881, ii, 529.

It is not during exposure to the abnormal conditions, but on returning to the normal atmospheric pressure, that the symptoms come on; often immediately, and always within half an hour or at most an hour of the return to the surface. The onset is usually preceded by other symptoms, and especially by pains in the ears and in the joints. The latter are very common, and occur after a much slighter degree of exposure than is needed to produce paralysis. They are felt chiefly in the larger joints, and may continue for days and even longer. Very rarely there is also swelling of the joints.

The deafness occasionally observed has been usually bilateral, often persisting in one ear, but passing away in the other. It has even been attended by severe vertigo. The severer degree has been ascribed to hæmorrhage into the labyrinth, but no pathological facts are at present available.

It is important to note the conditions under which the symptoms come on. Not only do those who are unused to such work seem especially liable to suffer, but there are also considerable individual variations in the degree of liability.

Paraplegia or hemiplegia may occur, but the former is far more common. The onset of the paralysis is sudden. The legs feel heavy and are found to be weak, and in a few minutes the patient is unable to move them. Sensation is often lost as well as motion, and in all severe cases the sphincters are affected. The arms are seldom involved. In slight cases the loss of power is incomplete, and one leg may be more affected than the other. The impairment of sensation is often imperfect or irregular. If the paralysis is incomplete the power may return in the course of a few days, but in severe cases the palsy usually lasts for weeks or even months, and it may be permanent. Death may occur at the end of a week or more, in the same way and from the same causes as in acute myelitis. When the paralysis is hemiplegic it is generally moderate in severity and transient, passing off in a few hours or days. It may be even still more brief, as in the case referred to above, in which a diver, after half an hour's work ninety-six feet below the surface, suddenly felt tingling across his loins, and that his right arm and leg were almost powerless; in half an hour he had recovered sufficiently to walk home. If cerebral symptoms are more intense, they are generally very severe with sudden loss of consciousness, continuing as deep coma, irregular breathing, and indications of cardiac paralysis. Such cases usually end in death in the course of a few hours.

Pathology.—The most probable explanation of these cases ascribes them to the escape from the blood of gases, with which it has become charged during the exposure to the high pressure. It is certain that a great excess of gas must be dissolved in the blood during the exposure, and that the amount, at least of oxygen and of carbonic acid, contained in the blood when the person emerges from the caisson must be vastly greater than normal. It must, moreover, be greater the longer the exposure, since the absorption of the excess will be a gradual process, taking place through the lungs in respiration, but it will be more rapid as the pressure is greater. These conditions agree with observed facts. After returning to the normal atmospheric pressure, the excess of gas probably passes off gradually by the lungs in most instances; but if extreme, gas may escape from the blood within the body. The occurrence of this has been proved experimentally (Hoppe-Seyler and P. Bert). Gas, so escaping, is no doubt quickly reabsorbed, but must exert a pressure capable of arresting the function of the structures of the nervous system. If abundant, it may conceivably rupture these. Further, the special effect on the nerve-centres may be connected with the position of these within cavities that are practically closed. These conditions, coupled with the extremely circuitous course of the blood from the cord, may explain the incidence of the effects on the nervous system.

The few ascertained facts harmonise with this pathology. Such escape of gas has been proved to result from diminution of the atmospheric pressure.* In many cases no visible lesions have been found, and it is obvious that gas, widely effused, may exert dangerous pressure, and yet may be speedily removed, so that no indication may remain of the cause that has completely arrested function.

It was once thought that the symptoms were due to local hæmorrhages, but extravasations are seldom met with, even in the results of experiment, and it is certain that they take but a trifling part in the production of symptoms. The only positive pathological observation agrees with the opinions above stated. Leyden† found (in a case of characteristic paraplegia) small irregular fissures in the mid-dorsal region, chiefly within the posterior and hinder parts of the lateral column. The fissures were filled with round-cells, but contained no red blood-corpuscles, and from their well-defined edges they were certainly not produced by the infiltration of the cells found within them. The only explanation that is satisfactory, or in any harmony with their features, is that they were produced by the sudden escape of gas, and were afterwards occupied by the round-cells.‡ The physical firmness of the cord is far less in the dorsal region than in the enlargements; and probably, as Leyden suggests, this is the reason why the dorsal region suffers structural damage from the escape of gas more than other parts. It is evident, however, that such escape may occur in the brain, for instance, even to a fatal degree, and leave no traces unless it occurs so rapidly as to cause laceration of the tissue. The absence of hæmorrhage of the ordinary character may be further explained by the fact that the effused air will resist the effusion of blood. Some other anomalous features, such as the transient character of the symptoms in some cases, are also explained by the peculiar nature of the lesion, which may exert a wide-spread and considerable influence, and in a short time may have vanished by the reabsorption of the compressing air. Hæmorrhage must be regarded as an accidental consequence, and the production of such fissures as were found by Leyden may not be a necessary part of the lesion, even in grave cases The mechanical effects of the gas may be considerable, and arrest the function of the nerve-elements, without the passage of the gas outside the capillary vessels.

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* Hoppe-Seyler, 'Müller's Archiv,' 1887; P. Bert, 'Comptes Rend.,' 1871–2.

† 'Arch. f. Psych.,' ix, Heft 2.

‡ The clinical condition of transient hemiplegia, which has been observed in empyema as occurring during irrigation, and thought by some to be the result of air or gas embolism, may be of similar nature.

The fact that the spinal cord suffers more than other organs may be partly due to the plexuses through which the blood can return only slowly to the lungs, where its relief from the surcharge of gas is effected. The same considerations apply to the brain. In both organs the escape of air is more instantly disastrous than elsewhere, and, indeed, may not only occur, but act in a peculiar way, on account of the position of the organs within chambers that are, to a large degree, closed. The conditions are too complex to permit us to follow them in detail, or analyse fully the mechanisms that determine the effect on the nerve-centres. The escape of the excess of gas in the lungs can only take place gradually, as successive quantities of the surcharged blood pass through the capillaries of the air-cells. If, in consequence of the slowness of the circulation in the cord, any escape of air occurs in its capillaries, the effect of this will be still further to hinder the local circulation, and to favour the further escape of gas. In this way we are able to discern something of the mechanism which determines the special affection of the spinal cord. The less the external support the less will be the resistance to the escape of gas, and the incidence of the lesion on the posterior part of the cord may be, in part at least, determined by the adoption of the recumbent posture when the first symptoms are perceived.

At the onset, when the first symptoms are experienced, it is probable that a return to a greater degree of pressure might arrest the development of the mischief. It is certain that, in all cases of exposure to a high degree of pressure, the transition to the normal pressure should be made gradually. When developed symptoms indicate that a definite lesion of the cord has occurred, the further treatment must be conducted on the same principles as in acute myelitis—the morbid process that has, in fact, been set up.

HÆMORRHAGE INTO THE SPINAL CORD; HÆMATOMYELIA.

Primary hæmorrhage into the spinal cord, sufficient to cause symptoms, is a very rare disease, and it is even more rare than is suggested by the cases now and then recorded as such. We have already seen that hæmorrhage may accompany myelitis; a considerable extravasation may occur when the inflammation is only commencing, during the state of congestion, and while the symptoms are slight. Such cases are easily mistaken for primary hæmorrhage. The risk of error is not always removed by pathological examination. Inflammation results from hæmorrhage, and when indications of myelitis are found about a clot, it may be impossible to say whether these are primary or secondary. It is probable that many cases of secondary myelitic hæmorrhage have been regarded and described as primary, and it is possible that a few cases of primary hæmorrhage have been regarded as secondary. One writer, Hayem,[*] goes so far as to deny the occurrence of primary non-traumatic hæmorrhage; but such an exclusive view is unwarranted. It is certain, however, that the history of primary hæmorrhage has been largely written from uncertain data, and will need extensive revision when a sufficient number of exact observations have accumulated. It is probable that the causal influences constitute the surest guide. When hæmorrhage occurs under conditions which usually induce myelitis, the extravasation must be regarded as the result of the vascular disturbance of commencing inflammation.

Etiology.—The rarity of hæmorrhage into the cord is especially great in comparison with the frequency of hæmorrhage into the brain. The difference probably depends on the tortuous and long course of the arterial path to the cord, whereby the vessels are preserved from the high pressure which is the chief cause of the degeneration, dilatation, and rupture of the cerebral arteries. Miliary aneurisms are not found within the spinal cord. Hæmorrhage is far more common in males than in females. It may occur at any age, and has been met with in young children, even so early as seven months,[†] while some of the subjects have been in advanced life; but it is most common between twenty and forty, *i. e.* during the first half of adult life. Many cases occur in young adults apart from injury or obvious exciting cause, and apart also from initial myelitis, which accounts, however, for some of the cases. It has been met with as a consequence of a hæmorrhagic tendency, as in a case in which it succeeded severe epistaxis, to which the patient, a young man of twenty-four, was liable.*

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[*] 'Des Hémorragies intra-rachidiennes,' Paris, 1872.

[†] Clifford Allbutt, 'Lancet,' 1870, vol. ii, p. 84. Numerous hæmorrhages were observed in the grey matter of the lumbar and cervical enlargements (probably polio-myelitic, but occurring the day after a fall) in a child of four by Chaffey ('Path. Trans.,' 1885).

Of immediate causes, injury is the most frequent, especially falls which involve a severe concussion of the spine; the spinal column may or may not be injured at the same time. Over-exertion and exposure to cold have in rare cases preceded the onset. Chronic alcoholism and sexual excess have been thought to predispose. In one case within my knowledge, an extensive hæmorrhage into the grey substance at the top of the lumbar enlargement resulted from coitus four times repeated, the symptoms commencing suddenly during the fourth act. Minute extravasations are often found after death from diseases which interfere with respiration and cause venous congestion, and they are especially frequent in maladies which at the same time cause functional excitement of the cord, as tetanus and all severe convulsions. They cause no symptoms, and are probably produced during the last moments of life. They have been termed "*accessory*." The diseases of the cord that lead to *secondary* hæmorrhage are chiefly inflammation, tumours, and cavities in the cord. The last-named condition is especially important. It is probable that hæmorrhage occurs more readily if the spinal cord contains a congenital cavity or fissure, due to an arrest of development, and surrounded by embryonal neuroglial tissue. Hence this condition will be found in disproportionate frequency in cases of hæmorrhage.

PATHOLOGICAL ANATOMY.—The minute extravasations just mentioned, as met with after asphyxial and convulsive diseases, are found in both the grey and white substance, but especially in the former. They are usually microscopic, or visible to the naked eye as minute red points, distinguishable from distended vessels only by their slighter resistance to a stream of water. The extravasation may occupy the perivascular sheath, or the cavity in which the vessel lies, or extend between the nerve-elements. The larger non-traumatic hæmorrhages, which cause symptoms, always begin in the grey substance, and are often confined to it, extending into the white columns only when large in size. The vessels of the grey substance are more numerous than those of the white, have less external support, and probably undergo more considerable changes in state. The effusion forms a cavity in the cord, sometimes rounded, sometimes irregular in transverse section, and half an inch or more in vertical extent. The cord is enlarged at the seat of the hæmorrhage, and this may be visible externally as a dark swelling the size of a nut or a bean. Very rarely the hæmorrhage tears the layer of cord which limits it, and blood, usually only in small quantity, escapes into the membranes. The tissue adjacent to the clot is usually broken down, stained, and softened, and inflammatory changes may be recognised in it with the microscope, just as in the neighbourhood of hæmorrhages into the brain. As in the latter, the effused blood slowly undergoes changes in tint, becoming first rusty and then yellow, and ultimately a cyst may remain. Several extravasations may co-exist, usually in the same part of the cord. In contrast to this *focal hæmorrhage* there may be an infiltration of the grey matter with punctiform extravasations, which may appear, until closely examined, to be a single hæmorrhage. The tissue between these small extravasations is broken down. It is probable that this form is always secondary to myelitis, as in the case mentioned in the foot-note on p. 437. In such secondary cases a careful microscopical examination usually reveals indications of inflammation much more extensive than the area affected by the hæmorrhage. A growth into which hæmorrhage occurs is usually a glioma, unaffected parts of which will be found in the neighbourhood of the extravasation. The cavities in the cord into which blood may escape are sometimes of considerable vertical extent. I have known a fissure in the posterior column to be filled with blood through almost the whole length of the cord. This condition thus favours both the occurrence and the extension of hæmorrhage.

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* Sinclair, 'Lancet,' 1885, ii, p. 1043.

SYMPTOMS.—Slight symptoms, "prodromata," have been observed in some cases, chiefly in the form of trifling sensory disturbance, tingling, &c., in the limbs afterwards paralysed. They have existed for a few hours or days, or even for two or three weeks before the onset. It is probable, however, that these have been cases of secondary myelitic hæmorrhage, and that there are no premonitory symptoms in primary extravasations. The actual onset is always sudden; the symptoms attain a considerable degree in the course of a few minutes, but they sometimes increase during one or two hours, when the hæmorrhage is from a small vessel and slowly increases in size—probably augmented by the rupture of other vessels which are torn in the tissue lacerated by the blood. Occasionally there has been transient loss of consciousness without any cerebral lesion, probably from the upward influence of the shock. Rarely the onset has been by a series of sudden augmentations of the symptoms. Sometimes the symptoms come on during sleep. The suddenness of the onset is the characteristic of the disease.

The symptoms which thus develop vary according to the seat and extent of the extravasation. In the majority of cases there is paraplegia, complete motor and sensory paralysis up to the level of the lesion, with loss of power over the sphincters. Pain commonly but not invariably accompanies the sudden palsy; it may be felt in the spine or in the sacrum, round the trunk, at the front of the thorax, or at the epigastrium, sometimes seeming to pass thence through the trunk to the spine, or it may be felt in the legs. If in the spine it is local, and does not extend through a considerable length of the spine, as in meningeal hæmorrhage, and there is not the initial spasm and rigidity which characterise the latter disease. This pain may precede the palsy, even for half an hour or an hour; it begins suddenly, and is no doubt due to the first slight extravasation, which afterwar increases (when some resistance yields), and effects the compressi that produces the paralysis. Thus a girl aged fifteen was seiz with sudden sharp pain, referred to the sternum at the mamma level, and to the corresponding region of the spine; this continue for half an hour, when the legs suddenly became powerless and inse sitive. It is highly probable that when there is such initial medi or bilateral pain the hæmorrhage occurs in the central region of th grey matter, perhaps from one of the commissural or anastomot arteries (see p. 227), and that the pain is due to the irritation of th fibres of the posterior commissure, in which the paths for pain cros the middle line. The spinal column may be tender opposite th affected spot. Usually the paralysed muscles are relaxed. Sometimes they are the seat of early clonic contractions, or these may come on a few days after the onset. When the hæmorrhage is in the cervical region all the limbs are powerless; one arm is often affected before the other. The state of reflex action varies according to the seat of the disease; if it is at first abolished it quickly returns in the legs (unless the hæmorrhage is in the lumbar enlargement), and it soon becomes excessive unless destroyed by secondary myelitis—a not unfrequent consequence. In one case there was a remarkable initial increase of myotatic irritability a few hours after the onset, probably irritative, and quickly giving place to loss.* Vaso-motor and trophic changes in the skin are common and often intense; there is often vascular dilatation; bedsores readily form, and cystitis may result. Frequently the secretion of sweat is increased for a time. The temperature is normal at the onset, but it generally rises in the course of a few days from secondary inflammation in the cord.

The palsy developed at the onset usually continues for a week or ten days, although the pain may lessen. The symptoms do not always increase during the stage of inflammation, perhaps because this only involves the structures which are already impaired by pressure; but if the patient has been brought near to death by the primary hæmorrhage, the secondary inflammation may end life. Occasionally, moreover, symptoms of an ascending or descending myelitis may come on, and the former may cause death by its interference with the muscles of respiration. This extension is greatest probably in cases of myelitic hæmorrhage, in which the extravasation is merely an incident in the course of a commencing inflammation, but it seems also to occur in cases of primary hæmorrhage. The slow extension upwards of the symptoms during the first week may be very distinct; and the occurrence of descending inflammation may be shown by the loss of reflex action, and by the failure of electric irritability in the muscles, sometimes only in those supplied from the upper part of the lumbar enlargement.

The symptoms pass into a chronic stage, improvement being usually slow. Some lasting loss of power remains in most cases, and there is often some permanent muscular wasting on account of the frequency with which the hæmorrhage is in the cervical or lumbar grey matter. Occasionally there is rapid recovery up to a certain point; a hæmorrhage of small size may abolish conduction in the white columns by the suddenness with which it compresses them, and the effects of the pressure may quickly pass away, while those remain that are due to the destruction of the grey matter.

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* Sinclair, loc. cit.

DIAGNOSIS.—The diagnosis rests on the actually sudden onset of the symptoms, and on the occurrence of pain, in a limited region, as part of these. The former is evidence of a vascular lesion (rupture or closure), and the latter shows an acute irritation of the nerve-elements, such as may be produced by their laceration, but not by mere deprivation of blood. It must be remembered, however, that we cannot assume that symptoms which come on during the night's sleep are of sudden onset. Neglect of this consideration sometimes causes a mistake in diagnosis. The mode of onset is a sufficient distinction from all other organic diseases, except hæmorrhagic myelitis and meningeal hæmorrhage. The former (really a variety of hæmorrhage) is distinguished by the existence of slight symptoms before the sudden attack. We are not justified in regarding as primary hæmorrhage any case in which premonitory symptoms existed for more than a few minutes, unless such symptoms were so pronounced and sudden in onset that they might have been due to a definite extravasation, afterwards increasing. Initial fever (within the first six hours) always makes myelitis probable, provided there is no other cause for it. The distinction from meningeal hæmorrhage has been mentioned in the account of the symptoms, and in the description of that disease.

PROGNOSIS.—In all cases in which the symptoms are considerable in degree or wide in range, the danger to life is great, and remains great until they begin to subside. The prognosis is better when the disease is in the dorsal region than when it is in the enlargements, for the same causes as influence the prognosis in myelitis. It is better when sensation returns in the course of a few days, but if the enlargements are affected other sources of danger remain considerable. Early trophic changes also render the prognosis worse, for they show an intensity of degree that may involve grave danger. After the onset is over, the forecast must be guided by the general principles that determine the prognosis in acute myelitis.

TREATMENT.—The treatment of hæmorrhage into the substance of the cord is the same as that of hæmorrhage into the membranes (p. 339) The measures requisite are few, simple, and all-important. Absolute rest and the prone position are to be secured before anything else is thought of. Ice should be applied to the spine over the seat of the hæmorrhage. The bowels should be opened freely, and full doses of ergot or ergotin may be given: ʒss of the liquid extract or five grains of ergotin may be given by the mouth, or three grains of ergotin may be injected under the skin, and the dose may be repeated two or three times, at intervals of two hours. A few large doses are probably more effectual than smaller doses continued for a longer time, since the hæmorrhage probably does not go on for long. The after-treatment must be that for myelitis. The disease is one of those in which most of the good that can be done by treatment—and in few diseases is the opportunity more urgent—rests with those in whose hands the patient is immediately after the onset.

DEGENERATIONS OF THE SPINAL CORD.

A large and important class of diseases of the spinal cord consists of those in which there is a slow degeneration of the nerve-elements, with an overgrowth of connective tissue, and in which structures are affected that have a common function, while others that have a different function escape even when they are adjacent to the elements that are diseased. Affecting thus functional "systems," they are termed "system diseases." This term has been used in several senses, more or less special (even based on the developmental relations of the structures), and hence the question whether a given malady is a "system disease" or not, is one to which various answers have been given. The term is here used in its widest and simplest sense, as meaning an affection of the structures that have a function either the same, or so far allied that they work together as parts of one system, distinguishable as such from other sets of structures. A "system disease" may involve all the structures of the system, or only some or one of them; its chief distinction is from a random disease that involves structures irrespective of function—merely, for instance, because they are contiguous, or supplied by the same artery.

In "system diseases" the primary change is, as a rule, in the nerve-elements, and the overgrowth of interstitial tissue is secondary. The process is analogous to that which occurs in the secondary degeneration of nerve-fibres, in which the first change is certainly in the nerve-elements, the destruction of which is followed by an overgrowth of nuclei and supporting tissue, amounting ultimately to a "sclerosis," as it is termed. The process is by some regarded as a chronic parenchymatous inflammation, an inflammation beginning in the proper functional elements of the organ, but it seems undesirable thus to widen and loosen our conception of inflammation; so far as the process is concerned the question is one of name rather than of nature.

Another question of much greater interest is the relation of the two elements in the process, the wasting and the growth, the atrophy of the nerve tissue, the hypertrophy of the connective tissue. The failure of nutrition in the one causes an increased energy of nutrition in the other. We have seen this relation in the nerve-fibres. The degeneration of a fibre is attended by an active growth of its nuclei and protoplasm. The nutrition of the two elements, the neural and the adventitial, is evidently connected in the closest manner, but in inverse course. It is important to recognise the fact that the process of growth of the connective-tissue elements is an active manifestation of nutritional energy, which, when once excited, may be to some extent independent of its cause. It may, when very active, pass beyond its proportional limits, and be greater than corresponds to the nerve atrophy that causes it, invading adjacent structures as if an independent process. It may, when very rapid, have some of the characters of an interstitial inflammation, and even an acute inflammation. Such excess is quite unusual; as a rule the secondary process is subordinate and proportioned to the primary change.

These degenerative diseases fall into certain types so far as the spinal cord is concerned, but they are subject to some variety of combination even there, and to great diversity of association with degenerative changes elsewhere. The affection of the cord is of either the sensory or the motor elements, or both. The type of the former is tabes; of the latter, either spastic paraplegia or muscular atrophy, according as the lower segment of the motor path is affected, or the upper. Other combinations of affection will be better understood when the diseases are described.

The degenerative diseases are not numerous, but they are of great importance. They are, degeneration of the anterior cornua and anterior root-fibres, causing muscular atrophy; degeneration of the posterior columns and posterior root-fibres, causing locomotor ataxy; and degeneration of the pyramidal tracts, causing spastic paraplegia, a disease the exact pathological position of which is not quite certain. These degenerations may be variously combined, and some combinations need separate description.

Their causes are various. In some the nutrition of the nerve-elements fails soon after they have achieved maturity, and this is prone to occur in members of the same family. In other cases, which present a contrast to those just mentioned, and yet are allied, there is a senile degeneration, apparently in consequence of less vital endurance in these elements of the nervous system than in the other structures of the body. In such cases the local degeneration merely anticipates the somatic death which it too often induces. In these we must assume an inherent deficiency of vital endurance in the structures that fail. A similar assumption seems to be the only explanation for the cases in which degeneration of certain elements of the central nervous system occurs in middle life, between thirty-five and fifty, usually as a result of depressing emotion, or of some influence which lessens the general nutrition. Lastly, we are confronted with a large and varied class of cases, in which similar degenerations seem to result from toxic agents present in the blood. These may be received from without, or generated within the system, and an especially potent source of such toxins seems to be the development of the organisms of some specific diseases.

LOCOMOTOR ATAXY; TABES DORSALIS.

The malady thus named is the most common chronic disease of the spinal cord. It consists in a degeneration in the posterior columns of the spinal cord, or the peripheral sensory nerves, or both. According to the current views, it is the expression of impaired nutritional influence of the sensory neurons of the spinal cord, primarily of those related to the afferent muscle-nerves, but often also other afferent nerve-systems, including those from the skin. The nutritional centre is in the posterior ganglia, and the toxin apparently acts first on the extremity of the sensory fibres, and also on those that pass into the cord. The effect is manifested, when considerable, by inco-ordination of movement, peculiar pains, defective sensibility, and loss of the myotatic irritability (muscle-reflex action), of which the knee-jerk is the most convenient manifestation. When slight in degree, the symptoms may be limited to the pains and the loss of the knee-jerk. The name "locomotor ataxy" was given to the disease by Duchenne; "tabes dorsalis," or "wasting of the back," is a term applied by Hippocrates to certain symptoms supposed to be due to venereal excess, and long ago limited in Germany to symptoms believed to depend on atrophy of the spinal cord. It then included all chronic paraplegias, but was further restricted to this disease by Romberg. It has lately obtained wider use on account of the discovery that inco-ordination may be absent when the disease is slight in degree.

The symptoms vary much in different cases. Besides the varieties thus produced, there are two allied affections that ought not to be classed with it. One is the so-called "hereditary ataxy." The other is that in which both weakness and inco-ordination co-exist from the first; "ataxic paraplegia" it may be called. Each differs from other varieties with sufficient constancy to merit separate consideration. These forms are not included in the following account.

HISTORY.—The inco-ordination of movement and other symptoms of the disease were frequently noted, during the first third of this century, in cases of disease of the spinal cord, but such cases were not distinguished from those with actual loss of power. The inco-ordination was found to be associated with disease of the posterior columns by Stanley.* The first really exact account of the disease was pub-

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* 'Med. Gazette,' Feb., 1840, and 'Med.-Chir. Trans.,' vol. xxiii.

lished in 1847 by Todd,* who distinguished the cases with inco-ordination and without weakness, from simple paraplegia, and, apparently not aware of Stanley's observation, he inferred (from the character of the symptoms and his theory that the posterior columns contain fibres connecting segments of the cord at different levels) that the posterior columns would be found diseased; and he verified this inference by finding in two cases disease of these columns. The credit of the discovery of the disease belongs, if to anyone, unquestionably to Todd, and few diseases can with greater truth be said to have been "discovered." Four years later (in 1851) Romberg described the disease and the lesion in the posterior columns, but he failed to exclude loss of power from the symptoms. Russell Reynolds, in 1855, gave an accurate description of the symptoms, and, in attributing the ataxy to muscular anæsthesia, was the first to give what we must now regard as the true explanation of the chief symptom.† A series of cases was described by Gull in 1856 and 1857. Türck first observed with the microscope the wasting of the fibres in the posterior columns. Duchenne in 1858–9 published an independent and very able analysis of the symptoms of the disease, and gave it the name "locomotor ataxy." He obtained for it (with the help of Trousseau's "Lectures") the recognition that previous descriptions had failed to secure, and achieved such a degree of success that the malady is called in France "Duchenne's disease." If any name is attached to it, that of Todd alone can be right.

Causes.—The disease is much more frequent, in this country at least, in urban than rural populations. Inherited influence is to be traced only in a small proportion of the cases, perhaps in not more than 10 per cent. It is usually a general neurotic heredity, manifested by such diseases as insanity, epilepsy, and other degenerative diseases of the nervous system. Instances of this are—father epileptic; father insane; two sisters insane. But in this small percentage the influence becomes insignificant in the presence of the chief cause, to be mentioned presently, since in many instances its coincidence must be accidental. Direct inheritance of the disease is extremely rare (the special hereditary form being always excluded). A boy with distinct symptoms and optic nerve atrophy was the son of a man who presented characteristic indications of the early stage of tabes (Remak). In most instances of this character the inheritance is not direct, being effected by means of syphilis. The inherited disease is probably the only cause of the juvenile form of tabes.

Males suffer far more frequently than females, the proportion being about ten to one, and this seems to imply some proclivity inherent in the male sex. A like preponderance of males obtains in a disease that has a close alliance with tabes—general paralysis of the insane. It is, however, not certain that the sexual difference may not be connected with the wider dissemination of the chief cause—syphilis—among men, and with some peculiar power of resistance to its influence on the part of women. The middle period of adult life is that in which locomotor ataxy usually commences. No less than half the cases begin between thirty and forty, one quarter between forty and fifty, and rather less than a quarter between twenty and thirty. It rarely begins after fifty, but I have once known it to develop at sixty-six. Under twenty it is still more rare, but cases are met with as early as ten, and even in quite young children, the subjects of the cause next described.

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* 'Cyclopædia of Anatomy and Physiology,' vol. iii, p. 721.

† Reynolds, 'Diagnosis of Diseases of the Brain, &c.,' 1855, "Anæsthesia Muscularis." Certain words are worth quoting:—"It appears most probable that the centripetal tract of fibres is affected, and that the locality of lesion is very variable" (p. 165).

Among the individual causes, one overshadows all the rest—the influence of syphilis. A very large proportion of the sufferers have had, at some previous time, constitutional syphilis, either distinct secondary symptoms or an indurated sore. The proportion is almost as large in the upper and middle classes as in the lower. Of 175 consecutive cases of the disease in men, seen in private practice (in which the past history can be relied on with more confidence than in hospital patients), no less than 114 of the cases, or 69 per cent., gave a history of a chancre known to be hard, or of secondary symptoms; and twenty-two others had had a venereal sore of unknown nature. The percentage of those with a history of syphilis or a hard chancre is 77. In another 13 per cent. there was a history of gonorrhœa, in some cases of repeated attacks; this involves exposure to the risk of syphilis. Patients with unquestionable secondary syphilis are often ignorant of any primary sore. When a disease is a distinct sequel of syphilis in so large a proportion of cases as 69 per cent., we are justified in assuming a similar relation in at least a majority of the cases in which infection cannot be excluded. Gonorrhœa emphasises the inability to exclude syphilis, but in no less than 3 per cent. of the total number of cases was there no exposure to the risk of contracting syphilis in the common way. In the lower classes the proportion is higher, and has been estimated at 80 per cent., or even more. The ascertainable facts are certainly below the real facts, as I have pointed out elsewhere.* When a deduction is made for possible accidental coincidence, there remains at least one half of the cases in which numerical coincidence must depend on causal relationship. It is probable, indeed, that, taking all cases, a causal proportion of three quarters would be nearer the truth. In women also antecedent syphilis can often be traced, although rather less frequently than in men. At the same time the facts are even more difficult to ascertain, since in married women syphilis so often runs a latent course. But in the cases of tabes that succeed syphilis the lesion is not syphilitic in histological character; it is as degenerative as in the cases in which syphilis can be excluded, and it is not influenced by the treatment for syphilis. Hence it must be regarded as a degenerative sequel of syphilis rather than as a true syphilitic disease. It is probably the influence of syphilis that determines the greater incidence on the urban than on the rural population, and the preponderance of cases in middle life. Inherited syphilis is also capable of causing the disease, and is to be traced (as far as I have seen) in all cases in children. After the acquired disease the interval between primary syphilis and the first symptoms of tabes varies from one to twenty years. It is, however, rarely less than three years, and in most cases it is between six and twelve years. In rare cases the first symptoms occur during the active stage of syphilis.[*]

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* 'Lettsomian Lectures,' 1889.

It is probable, however, that syphilis is not the only cause of the disease. In a few cases, less than 10 per cent. of the whole, it can be excluded with confidence. The causes operative in these can be traced also in some of the patients who present a history of syphilis, and in such cases the causation of the malady is probably complex. Hitzig and Buzzard have suggested that a toxin having a similar degenerative action may arise from soft sores.

One of these causes, which can sometimes be clearly traced, is injury, such as involves concussion of the spine. The immediate results of the injury, whether slight or grave, transient or lasting, are followed by the symptoms of the degenerative malady. In one case a man fell from a height on to the deck of a ship; transient paraplegia resulted, but six weeks after the accident the man presented extreme ataxy, with good power, and no knee-jerk (Arnold). I have known the symptoms to develop gradually a few months after a fall from a horse. Exposure to cold and wet has occasionally preceded the onset so directly that it must be regarded as, at least, an exciting cause. There is a history of syphilis in the majority of cases in which the manifestation of tabes follows some immediate cause. In other cases the disease has succeeded excessive fatigue and over-exertion, and also certain acute diseases, especially (it is said) acute rheumatism and typhoid fever; it perhaps follows diphtheria in rare cases, but most cases have been examples of the ataxic form of diphtheritic paralysis, ultimately passing away. Alcoholic excess has been noted in some instances, but most recorded instances have been due to peripheral neuritis, involving chiefly the afferent muscle-nerves. Sexual excess has been supposed by some to be a cause; but its influence can rarely be detected, and its significance is uncertain, since sexual excitement is undoubtedly sometimes an early symptom of the disease.

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[*] Although an occasional relation to syphilis had been noted by several preceding observers, Fournier was the first (in 1876) to assert the wide extent of this relation. His statements were received with doubt (because syphilitic patients constituted his field for observation), but they were confirmed from the neurological side by myself ('British Med. Journal,' March 1, 1879) and Erb ('Arch. f. klin. Med.,' July, 1879); while abundant corroboration has been since afforded. Many who at first doubted have been convinced by fresh observations. Facts collected without reference to any point at issue are generally worthless, and freshly collected facts have thrown a new light on the subject. The proportion of cases with previous syphilis necessarily varies according to the absolute frequency of syphilis. The difference in the character of the lesion from that of changes known to be syphilitic was urged as an objection, but such considerations are theoretical, and must yield to facts; instead of denying that this or that lesion can be produced by a given cause, we may have to widen our view of the operation of that cause. Other degenerations of the nervous system seem also to have a relation to syphilis, as I pointed out some years ago ('Lancet,' Jan. 15, 1881). As will be shown in the section on Pathology, recent discoveries lessen very much the difficulty of comprehending the relationship by demonstrating the profound influence on the nervous system of chemical toxins generated by the organisms which we have learned to discern as the *materies morbi* of specific diseases.

Secondary Tabes.—Symptoms of locomotor ataxy sometimes succeed other diseases of the spinal cord, and such sequence is especially common in syphilitic subjects. Myelitis and syphilitic gummata may be thus succeeded by tabes. An officer in India, who had had syphilis, having suffered for a day or two from pains in the back, took a bath in snow water, and in a few days his legs were absolutely powerless. He gradually recovered power, but could not co-ordinate the movement: as power returned, lightning pains came on, and a year afterwards he presented the typical condition of locomotor ataxy. Again, a man, twelve years after syphilis, had a severe fall, followed by gradual loss of power, so that at the end of three weeks he could scarcely stand. The legs remained weak for a month, and then improved, but ataxy came on; three months later, power was good, inco-ordination extreme. In many cases, however, the ultimate condition is one of combined weakness and ataxy.

Symptoms.—A typical case of developed tabes presents certain motor, sensory, and reflex symptoms. There is inco-ordination of movement of the legs, sometimes of the arms also, without loss of power or muscular wasting. There are pains in the affected parts, especially sharp momentary "lightning" pains; there is some loss of sensation; there is often loss or diminution of reflex action from the skin, and almost always entire loss of the myotatic irritability that is revealed by the so-called "tendon-reflexes," and especially by the knee-jerk; there may be retention or incontinence of urine, constipation, and often loss of sexual power. Of this group of symptoms, two usually precede the others—the pains, and the loss of the knee-jerk. These may exist alone, even for years, before inco-ordination comes on. Thus the symptoms are far wider in range than the name "ataxy" suggests; and while inco-ordination, if it exists, is the most obtrusive objective symptom, it may never be developed. Hence physicians have extensively fallen back on the older term "tabes dorsalis." But recent expansion of our knowledge, especially the discovery of the extent to which the symptoms may depend on disease of the peripheral nerves, shows that even the qualifying "dorsalis" narrows the term unduly, and "peripheral neuro-tabes" has been added to the terminology of the disease.

Besides the symptoms above enumerated, others are occasionally present. Of these the most important are atrophy of the optic nerve (and occasionally of other cranial nerves); trophic changes in the skin, the bones, and the joints; peculiar paroxysmal visceral disturbances and occasionally motor paralyses and muscular wasting. The combinations of symptoms present in different cases vary much. It will be convenient to consider first the individual symptoms in their various degrees, and then their grouping and sequence.

Motor Symptoms.—The characteristic inco-ordination of movement develops gradually. It is usually increased (as Romberg pointed out) by closure of the eyes, and at first may only exist when the guiding influence of vision is thus withdrawn. Before it causes ataxy of movement it may render difficult the maintenance of equilibrium when the base of support is narrowed by the feet being placed close together, toes and heels; if then the eyes are closed the patient sways, and tends to fall. The effect of closure of the eyes is greatest when sensation in the soles of the feet is defective, but does not depend on this defect; it may be marked when sensation on the soles of the feet is perfect. The early defect in co-ordination may be discovered by the patient when he walks in the dark or backwards, or, not uncommonly, when he shuts his eyes in the process of washing the face. In a further degree of inco-ordination there is inability to stand with the feet together, even when the eyes are open, and the patient is only steady when the feet are wide apart. If the feet are bare, the difficulty is greater, because muscular action has to replace the rigid base of the boot. The irregular contraction of the muscles is shown by the conspicuous movement of the tendons on the back of the feet. The patient may oscillate from toes to heels before he comes to rest. As the defect progresses, uncertainty is felt in walking even with full visual guidance, especially on uneven ground, or on a very smooth surface. A slight visible alteration in gait is then appreciable, the feet are not placed on the ground quite as in health, or there is distinct difficulty in maintaining equilibrium when the patient turns quickly, and he has to put a foot down suddenly to keep from falling. As the inco-ordination increases, the change in gait becomes greater, but varies much in its precise characters, according to the muscles that are most affected. Often the feet are raised too high, thrown forwards too far, brought down too suddenly, and the whole sole comes in contact with the ground at once. Often the foot becomes inverted when it touches the ground. Efforts to correct error in movement have themselves to be corrected. In other cases the defect in maintaining equilibrium is greater than the actual disorder of movement of the legs, and the patient sways about in the manner of one who has cerebellar disease; this probably depends on a preponderant affection of the muscles of the hip-joint.

As the defect progresses, the patient is only able to walk by steadying himself with a stick, or by taking hold of the arm of another person or of adjacent objects. At first a very slight degree of this help is sufficient; guidance rather than support is needed. Afterwards, however, considerable support is necessary, and ultimately the patient may be unable to stand even with help. When he attempts to rise, the legs move hurriedly forwards and backwards, and if the upright posture is at last achieved, the legs slip forward, and only strong support saves the patient from a fall. The ataxy is manifest also in other movements of the legs. If the patient, when lying, tries to touch an object with his foot, the leg is moved irregularly, goes beyond the place, and then is brought too far back, and only at last does it come in contact with the object, often with unintended force. This inco-ordination, like that in standing, is much greater if the eyes are closed.

The arms may present similar inco-ordination, although they often escape, even when the affection of the legs is extreme; very rarely the arms suffer before the legs (brachial tabes). In one such case there was loss of sensation to touch, not only in the arms, but on the trunk between the epigastric level and the distribution of the cervical plexus. The commencing defect is revealed by delicate movements, such as writing. When slight, it may be conspicuous if the patient tries, with closed eyes, to touch some object, such as his own nose, or, having abducted his arms, tries to bring the forefingers together. As it increases, all movements become irregular; it is impossible for the patient to button his coat, or to pick up a small object from the table; the fingers twist about in the attempt. The grasp is not sustained; first one finger is felt to relax and then another. If the patient attempts to hold out his hand in a fixed posture, it is seen that the same irregularity obtains; instead of a uniform balanced contraction the muscles contract and relax involuntarily, and slow unintended movements of the fingers result, sometimes closely resembling those of athetosis. The same spontaneous movements may also be observed in the legs. They cease at once when the muscular effort is relinquished. Occasionally the muscles of the trunk present inco-ordination. Thus one patient could sit steadily on a chair when his eyes were open, but if he closed them would at once fall off. The movements of the head, face, tongue, and eyes escape the characteristic derangement.

Even with extreme inco-ordination, the power of the muscles may be unimpaired. Occasionally some group of muscles, as the flexors of the ankle, become weak for a little time and then strong again, just as may the eyeball muscles, as we shall presently see. In some cases motor power in the limbs remains unimpaired to the end; more often, when the ataxy has become great, some muscular weakness supervenes, with or without wasting of the weak muscles. There is a distinct group of cases in which weakness and ataxy come on together, but these are considered separately ("Ataxic Paraplegia").

Sensory symptoms are prominent in most cases,—subjective sensations, especially of pain, and loss of sensibility. Spontaneous pains are present in some degree in nine tenths of the cases of tabes. The most frequent and characteristic are the sudden and lancinating pains called "lightning pains." They occur chiefly in the legs, but may be felt in the trunk, arms, and even in the head and face. They are usually paroxysmal, and apt to come on at night; attacks of such pains last for some hours or for a day or two, varying in seat, but often felt in the same part throughout an attack, or through many attacks. Sometimes they are felt in a limited area of the skin; at others they seem more deeply seated; sometimes they dart down the limb. They may correspond to a nerve-trunk (especially the sciatic), but more frequently have no relation to the nerves. They may be felt near joints, but are seldom referred to them. Although the pains are usually acute and "stabbing," they are sometimes of different character. They may be compared to bad toothache, even when lasting only a second or two. In one patient the sensation was as if both legs were for a moment on fire. The pains in the trunk sometimes correspond in seat to a zone of hyperæsthesia, especially when there is a marked "girdle pain." When the pains are referred to the skin, this often becomes tender after the pains have been felt for a time; the contact of the bedclothes cannot be borne, and even a breath of air can scarcely be endured. Curiously, this may be the case when there is impaired sensibility to pain, but not to touch. When the pain is in the face it may closely simulate trigeminal "tic;" only it is on both sides, and such symmetrical facial neuralgia is very suggestive of a tabetic origin.

When pains continue at one spot for some days, vascular or trophic changes may occur there,—ecchymoses have been observed, and I have known the growth of the hair to be changed where pains had been felt in the scalp; each hair bent and broke off near the skin, over an area the size of half a crown; after the pains ceased the growth of the hair became normal. A sharp pain may be accompanied by a sudden reflex spasmodic movement of the legs, or by inhibitory weakness. Thus, in one case, a sudden pain would often make the patient fall on his knees. The pains may be so severe as to prevent sleep.

Other kinds of severe pain, less brief than the lightning pains, occur in some cases, and are described by various epithets, as "burning," "tearing," "gnawing." Burning pain in the toes distressed one patient. Much more common, and occurring with other pains, are slighter dull pains like those of rheumatism, for which they are often mistaken, an error that is facilitated by the circumstance that any of the pains of tabes may be influenced by weather, being especially increased by damp cold. Indeed, this occurrence on any sudden change in weather is often a marked feature. It is not uncommon for a patient to describe them as "equivalent" to a barometer. They may also be excited by indigestion. Dull pains sometimes precede the sharp pains, occasionally for years. A painful sense of constriction "girdle-pain," is also common, and may be felt around the trunk or in the legs, groins, &c.; it is sometimes referred to a considerable area—

the patient may feel as if the whole trunk were tightly enveloped in a cuirass of metal. Darting pains also may be felt in the trunk and not in the legs. Such a sense of constriction is sometimes referred to the legs, especially to the knees, but sometimes to the feet. One patient described this sensation as if an iron band were around each foot. In the arms and hands the pains are usually slight in degree, and seldom cause real distress; they are often felt chiefly in the ulnar region. Visceral pains may also occur, usually paroxysmal, and referred to the stomach, bladder, rectum, &c.

Pains are not only the most common, they are often the earliest indication of the disease, and may be the only subjective symptoms in slight and stationary cases. They bear no proportion to the other symptoms, and are sometimes trifling—occasionally altogether absent. They seem sometimes to occur alone; I have seen several patients who suffered lightning pains, characteristic in features, course, and general associations, both with and without the pupil symptoms common in tabes, but with no loss of the knee-jerk. The pains are then usually superficial, and the cases may be termed "cutaneous neuro-tabes." Ataxy may supervene, with loss of the knee-jerk.

Sensations other than pain are also common. They are various in character, described as "tingling," "pins and needles," "creeping," and very often as if the skin, especially of the soles, were covered by some soft substance. Sensations of cold or of heat are also common; the feet may feel as if the legs are always in cold water; a sensation of cold about the testicles is occasionally complained of. Similar cutaneous sensations are also common in the hands. They are especially common in the distribution of the ulnar nerve. Here also there may be impaired sensibility, especially for pain and temperature, when the sensory condition elsewhere in the arm is normal. The ulnar nerve has also been found insensitive to pressure (Biernacki). Increased sensitiveness to pain may also exist, especially on the soles of the feet, and often accompanies lessened sensibility to touch.

A diminution of sensibility is also present in most cases of the developed disease. It may involve all forms of sensibility, but seldom equally; some forms may be impaired and others normal. Either pain or touch may be affected alone; the most frequent change is loss of sensitiveness to pain. Delay of the perception of painful stimuli is a common early symptom, as will presently be described. A firm touch (*i. e.* slight pressure) may be felt when a slight touch is unperceived. When a touch is felt, the power of localising it may be impaired. Temperature sensations are rarely affected without other forms of sensibility, but they are often impaired with that of pain when tactile sensibility is normal. On the other hand, there may be extensive loss to pain without any defect of the temperature sense. A prick may then cause only a sensation of heat or burning, and extreme degrees of heat or cold cause pain less readily than normal.

The temperature loss may be partial,—either cold or heat may be unperceived while the other is recognised. When touch is slightly impaired, there may be an inability to discriminate differences in the degree of pressure on the skin.

Accompanying the diminution or increase of sensitiveness there are sometimes curious changes in the sensation produced. One of these is a delay in the perception of pain, which may amount to several seconds. The greatest delay I have met with amounted to seven seconds, but one of fifteen seconds has been described (Eulenberg). The contact of a point may be felt at once, the pain only after a considerable interval. When felt it may be excessive in degree. Often there is an after-pain, lasting sometimes for a quarter or half a minute or longer, and the maximum sensation may not be attained until several seconds after the pain is first felt. Thus, in a case in which there was a delay of seven seconds, the maximum sensation was only felt twenty-five seconds after the prick (Obersteiner). Often there is a second or third maximum, a sort of rhythmical recurrence of sensation, quick or deliberate. A delay greater than normal may also attend the sensation of heat, but this is less easy to recognise on account of the time normally required to raise the temperature of the skin. The localising power is sometimes strangely perverted. A touch on the toe may be referred to the back of the foot even when there is no ataxy. A touch or prick on one leg may be referred to the other, sometimes with singular uniformity in position ("allocheiria," p. 14). A prick in one spot may be felt in many places ("polyæsthesia") on the same, or on both legs. There may also be impairment on the face corresponding roughly to divisions of the fifth nerve. Occasionally a patient feels as if the face were covered by a mask, stretching and compressing the skin, a symptom named by Charcot "Hutchinson's mask." The reaction of the sensory nerves to electricity may be changed in the same way as that of the motor nerves when they are degenerated; instead of the earliest sensation, on closure of the circuit, occurring at the kathode, it occurs at the anode (Mendelssohn).

Impairment of sensation is a very variable symptom. It may be absent even when the spontaneous pains are severe. It is most frequent, and usually greatest, in the lower part of the legs and feet. It may even be limited to the soles, and usually then ceases at the outer sharp edge of the foot, but extends for an inch or so up the inner side. Touch or pain may be lost on the sole only, and even, strange to say, the form of sensibility that is lost on the sole may alone persist in the legs. Although the sole is usually most affected, sensation may be perfect on the sole when lost elsewhere. One leg is often more affected than the other. The loss may extend to the trunk, and over part or the whole of it. Defective sensibility to touch is sometimes met with around the anus, perinæum, scrotum, &c., and sexual loss is said to be usually associated with loss of sensibility in

penis.* The characteristic pain which pressure on the tes-
is frequently absent. In the upper limbs the impair-
n is usually greatest on the hands; it often commences
aspect of the fingers, and may be limited to this
hands, just as it is to the soles of the feet. Sensation
the trunk and not on the limbs—an important fact,
anæsthesia may then readily be overlooked. The impair-
trunk frequently ceases when the level of the third rib i
On the head, loss of sensation is rare and usually partia
defect of sensitiveness to pain over the whole hea
met with.

sensibility is not confined to the skin, but affects t
also in many cases. The contraction of the muscl
stimulation may be unfelt, and the muscles may be le
normal to pressure or forcible extension. It is on
patients that this can be distinctly ascertained; b
defect is probably an early change. In a case in whi
was much greater in one leg than the other, in t
the muscles were insensitive to traction, which excit
on the other side. The muscles may indeed b
when cutaneous sensibility is little or not at a
It is probable that the sensibility of the joints, fibro
is also involved. The sense of posture may be lo
sensibility is intact. The anæsthesia may extend to t
the trunk is involved. In consequence of the loss
injuries to the limbs, such as burns, may be unp
disease may be unattended with the customa
for instance, may be absolutely painless.

of sensation is not invariable. There may be
who present considerable inco-ordination, and
severe pains, as well as in those who have had n
to have no sensory defect when inco-ordinatio

from the skin is usually impaired in proportion to
sensibility, and especially to the loss of tactile se
be normal when pain is unfelt. The plantar re
impaired, and the progress upwards of the dise
by a progressive loss of the gluteal, cremasteric, a
On account of the variations met with in hea
whether reflex action is ever lessened when there is
sensation. On the other hand, in the early stage
is sometimes a considerable and even extreme exce
reflex action.

power is an exceedingly common and often an early
the disease, and may precede any impairment of sensory
As has already been stated, the loss is said as

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* Brown, 'Lancet,' 1898.

a rule to be accompanied by loss of sensation in the glans penis. Yet in such cases nocturnal emissions may occur, sometimes very frequently. On the other hand, exceptions are met with in which sexual power persists to a late period of the disease, even until after the patient has lost the ability to walk alone.

If the condition of cutaneous reflex action is somewhat inconstant, this is not the case with the reflex process on which the myotatic irritability depends. A loss of the knee-jerk, as Westphal first pointed out, is one of the earliest and most constant symptoms of tabes. With it disappear also all other indications of this irritability. In cases with great excess of cutaneous reflex action the knee-jerk is lost as in other cases, and the contrast between the two forms of reflex action is very striking. It is exceedingly rare for the knee-jerk to be obtained in any case of true tabes, but an early case is sometimes met with in which it is not lost, although diminished, and commonly unequal on the two sides. In one or two cases I have found it slight on one side and lost on the other when distinct ataxy was present, and have observed its subsequent disappearance.* In one case, in which the jerk was lost in one leg only, lightning pains were confined to this leg. Very rarely the knee-jerk persists and is equal, although there are characteristic pains, loss of the iris reflex, and a history of syphilis. The loss often precedes for many years the development of incoordination. It is important to remember that a true reflex action may simulate the knee-jerk when it is lost. It is distinguished by the conspicuous interval between the blow and the movement, by the wide range of this, and by the fact that a similar effect can be produced by a prick on the skin over the patellar tendon.

Eye Symptoms.—The functions of the internal muscles of the eye are often affected in tabes. In more than five sixths of the cases † the reflex action of the iris to light is lost; rarely the action remains perfect throughout. In most cases of reflex loss the contraction on accommodation is preserved, as first noted by Argyll Robertson. Often also (as Erb has shown) the dilatation that occurs on painful stimulation of the skin of the neck, &c., can no longer be produced. Sometimes the ciliary muscle is also paralysed, causing loss of accommodation. Very rarely there is loss of accommodation and of the associated contraction of the iris, without loss of the light-reflex. When reflex contraction is not lost it may not be maintained; the pupil again enlarges; an actual inverted reaction to light and darkness has been observed.‡ The pupils are often very small ("spinal myosis"), especially when there is loss of the reflex dilatation from the skin, a loss which seems to be accompanied by atony of the radiating fibres supplied by the sympathetic. The pupils are not always small; they may be of a

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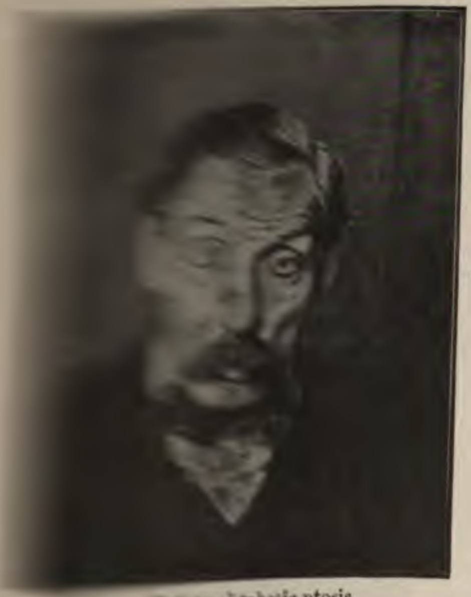
* Its return on the paralysed side in a tabetic patient who became hemiplegic, has been recorded (Hughlings Jackson and James Taylor, 'Brit. Med. Journ.,' vol. i, 1894).

† Berger says 97 per cent.

‡ L'Abundo, 'La Psychiatria,' 1889, vii, p. 286.

[illegible] still larger, and then I have usually found that t [illegible] obtained, although the light-reflex cannot. Th [illegible] circular, and are frequently unequal. Both ey [illegible] the same manner; occasionally the reflex actio [illegible] only lessened in the other; this is true also o [illegible], almost every possible condition is occasionall [illegible] other hand, the intra-ocular muscles may be [illegible] advanced cases. According to Berger the ocular [illegible].

[illegible] external ocular muscles is also common in tabes, [illegible] forms. (1) Transient weakness lasting a few



[illegible] tabetic ptosis.

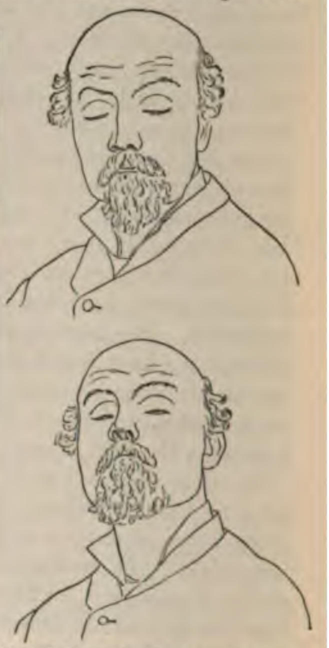


Fig. 120.—Double tabetic ptosis.*

[illegible] and then passing away. (2) Permanent paralysis, [illegible] of a single nerve or part of a nerve. Either [illegible] any stage, but the first is most common in the [illegible] second in the later stages of the disease. The transient [illegible] diplopia a common symptom in the early period; any [illegible] affected, but the external rectus is that which most [illegible]. Transient ptosis may precede an almost complete [illegible] ophthalmoplegia. The persistent palsy may affect one or more [illegible] the levator is involved, causing what has been termed [illegible]" (Figs. 119 and 120). Sometimes the whole third nerve [illegible] (3) There may be a combined palsy, suggesting a central

[illegible] Salpêtrière photograph, for which I am indebted to M. Charcot.

origin—as, for instance, loss of the movement of convergence associated with loss of accommodation, although the internal recti act in the movement towards one side. (4) Many or all the muscles of both eyes may gradually become paralysed—"progressive ophthalmoplegia," internal or external, a condition that is more fully described in the account of ocular palsies in Vol. II.

Atrophy of the optic nerve is the most serious ocular complication of tabes. Its frequency is difficult to ascertain, but is certainly less than is often supposed; it does not occur in more than one case in ten. Atrophy is generally an early symptom, usually commencing before inco-ordination is developed, and in a large number of cases ataxy does not supervene; if it does, the arms are frequently affected before the legs. There seems a tendency for the spinal malady to become stationary when the optic nerve suffers. The disc becomes pale, and its substance shrinks, so that recession begins at its edge instead of at the central "cup," as may readily be perceived by the course of the vessels. The failure of sight usually commences with a peripheral limitation of the field and loss of colour vision, but sometimes central acuity fails early. When this remains good, the peripheral loss may reach a considerable degree before it is suspected by the patient. Rarely there is a symmetrical sectorial defect in the fields. The visible pallor of the optic disc is not always proportioned to the failure of sight; the change in tint depends on wasting of the capillaries, and this does not always correspond to the wasting of the nerve-fibres. The ultimate tint is grey, or a whitish grey, especially as seen by the direct method. When the disc was very vascular before the atrophy, the grey tint is generally deepest. The vessels are but little narrowed. Occasionally there are slight signs of congestion in the early stage, and then some tissue of gelatinous aspect may develop in the disc, and the vessels may appear somewhat compressed.*

The course of the atrophy is usually slowly progressive, and in most cases it ends in total or almost total blindness. It may, however, cease to progress, and even undergo slight improvement. The onset is not always gradual; sometimes sight fails considerably in the course of a few days, without ophthalmoscopic changes to account for it. I have met with one such case in which double temporal hemianopia indicated disease at the optic chiasma. In such cases the secondary interstitial process probably assumes inflammatory intensity (see p. 443). The atrophy is often more advanced in one eye than in the other, and very rarely one eye only suffers. Flashes of light are occasionally experienced in the course of the atrophy, comparable to the lightning pains in the limbs, but they are not common.

Deafness, having the characters of nerve-deafness, is met with in some cases, sudden or gradual in onset, transient or lasting. The persistent deafness has been attributed to an atrophy of the auditory nerve, analogous to that of the optic nerve, but certainly in many cases without sufficient reason.[*] Only when there is a progressive limitation of the range of hearing, analogous to the peripheral limitation of the field of vision, are we justified in assuming nerve atrophy. I have seen two cases of this kind. In each there was also optic nerve atrophy, and the other symptoms of tabes were distinct.[†] Attacks of vertigo are frequent in the cases attended by deafness, and seem to depend on the disturbance of the labyrinth or auditory nerve, and to be thus connected only indirectly with the primary disease. But chronic slight vertigo from this cause increases very much the tabetic unsteadiness. Anosmia occasionally occurs, and is probably not infrequent. It is apparently due to an atrophy of the olfactory nerve similar to that of the optic.

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* Further details will be found in 'Medical Ophthalmoscopy.'

The functions of the other cranial nerves are not often affected. Pains may be felt in the region of the fifth nerve, and there is occasionally loss of sensation in some part of the area supplied by it, on one side or both. Unilateral atrophy of the tongue has been sometimes noted. In the larynx, besides the spasm to be presently described, paralysis of the vocal cords is met with, both as an isolated symptom and also after attacks of spasm have occurred for a long time. Both posterior crico-arytænoids may be paralysed, and in one case paralysis of one posticus occurred very early in the disease (Remak).

Sphincters.—The functions of the bladder are frequently deranged; there may be either sluggish micturition or a tendency to incontinence, and very often the bladder is not perfectly emptied. These symptoms frequently occur early in the disease, and it may be indeed only for them that the patient seeks medical advice. The retention may become absolute, or there may be overflow incontinence; there is rarely paralytic incontinence. The imperfect contraction, even when slight, may have serious effects. The residual urine tends to decompose, and gradually induces over-distension; cystitis is apt to occur, and secondary renal disease may develop insidiously, and is a not uncommon cause of death when the difficulty in micturition has been insignificant. Slight intermittent febrile disturbance is a frequent consequence of this bladder incompetence; it may cease entirely if the catheter is regularly used. The sphincter ani is often also weak, so that a loose stool can be retained with difficulty; but its paralysis rarely reaches a high degree. Constipation is extremely common.

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[*] It is very difficult to distinguish an affection of the nerve from one of the labyrinth independent of the nerve. Even in a case of bilateral deafness coming on in the course of tabes, in which the patient could hear only a loud voice, and deep notes better than high ones, in which atrophy had been diagnosed, Lucae found calcareous masses in each of the labyrinths, and the auditory nerves were quite normal.

[†] In one, only notes between E^2 and D could be heard. In the other the patient, when first examined, was absolutely deaf to notes above E^2 and below G_1. A few months later the restriction had confined the range of hearing to the octave in the treble clef between E^2 and E^1, even Eb^1 being inaudible. Thus the loss occurred chiefly from below upwards. Ultimately no note could be heard.

Vaso-motor and trophic disturbances constitute an important group of symptoms, and may reach a considerable degree without a coincident increase in the other symptoms. Local sweating has been noted, confined, for instance, to the palms and soles, or to one side of the head. The ecchymoses and altered growth of hair, in connection with attacks of pain, have been already mentioned. Pigment may disappear from the skin and hair in isolated spots. Herpes of the skin is not uncommon, and usually occurs in association with attacks of pain. A peculiar association was present in one case; attacks of limited scrotal pain were accompanied by a "rash" on the foreskin. The epidermis of the sole becomes thickened; blisters readily form beneath it, and may lead to indolent ulcers. The curious affection known as "perforating ulcer of the foot" has been found to be almost confined to cases of tabes. Troublesome ulceration of the toes, especially about the nails, is also occasionally met with, and may necessitate amputation. The growth of the nails of the feet, and sometimes of those of the hands, may be changed; they may be thickened, and the surface furrowed or irregular. The nails may even fall off and be slowly renewed. The teeth sometimes decay quickly, and may drop out. One patient, who had previously lost many of his nails, found that all the teeth of the upper jaw fell out in the course of three days, without any pain or decay. Wasting of the muscles occurs only as a complication in the later stage of the disease.

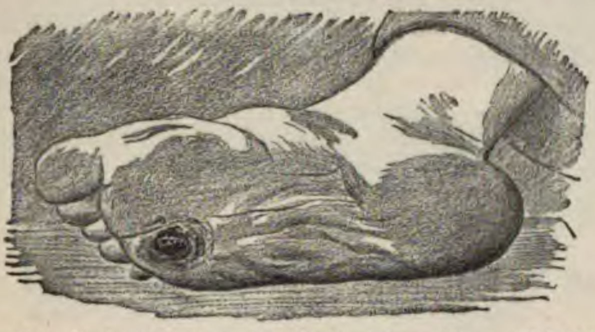

FIG. 121.—Perforating ulcer of the foot in tabes. (From a Salpêtrière photograph.)

Changes in the nutrition of the joints and bones have attracted much notice since attention was directed to them by the careful investigations of Charcot, whose name is sometimes connected with the condition. They are not very common, but are sufficiently frequent and well marked to place their relation to the disease beyond doubt. The bones may become brittle, and may break readily, in so-called "spontaneous" fractures. The process of union is attended with the formation of a large amount of callus. Sometimes there is also ossification or calcification of the structures adjacent to injured bones, and even of the fibrous tissues independent of the joints, such as the intermuscular and subcutaneous tissues. These may be rapidly inflamed, and extremely quick swelling may occur, followed by induration. As the inflammation subsides, there is a remarkable tendency for ossification to occur even in the newly formed inflammatory tissue beneath the skin. Such subcutaneous changes occur most frequently in the foot or thigh, especially near joints, but sometimes in the deeper fibrous tissues between the muscles. In the joints the changes consist of erosion of the cartilage, wasting of the heads of the bones, and ossification of the ligaments; sometimes irregular bony growths form, and occasionally the head or shaft of a long bone may undergo an extraordinary increase in size by the development of new osseous tissue on the surface. The joint changes may thus assume an atrophic or hypertrophic character, and the movement may be either too free or too limited. Wasting of the head of the femur often renders dislocation of the hip easy. With such changes, and often when they are slight in degree, there may be great effusion within the joints and œdema outside them. The larger joints are those most commonly diseased—knee, hip, ankle, elbow,—but the small joints of the fingers have been known to suffer (Westphal). The relative frequency with which the chief joints are affected is approximately indicated by the following percentage: *—knee, 45; hip, 20; shoulder, 11; tarsus, 8; elbow, 5; ankle, 4. They often occur during the pre-ataxic stage. The lesions are sometimes excited by injury, and the extraordinary changes that may follow traumatic influences, or occur alone, afford a conclusive indication that abnormal trophic influences are at work. Fig. 122 represents the arm of a man who fell and struck his elbow, fracturing the olecranon and condyle. This was followed by extreme painless swelling, and bony masses could be felt, gradually increasing in size. After death the fractures of the bones were found, and masses of new bone had formed in the capsule of the joints. The arthropathic changes occur also, however, without

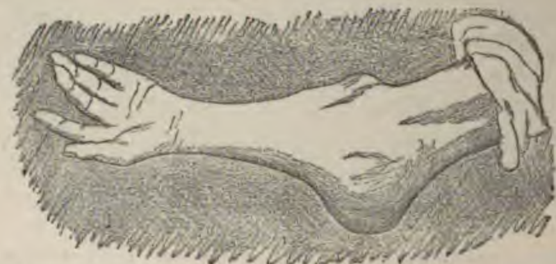

FIG. 122.—Locomotor ataxy, painless swelling of elbow-joint after a fall on it. One condyle of the humerus and olecranon were found, after death, to have been broken off. Several separate pieces of new bone had formed in the capsule of the distended joint.

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* Obtained by combining the collections of cases of Weizsäcker (Bruns' 'Beitr. z. kl. Chir.,' 1887) and Rotter ('Arch. f. kl. Chir.,' Bd. xxxvi)

injury, and are probably due to disease of the nerves, which not only has a direct trophic influence, but also renders the structures insensitive to the injurious effects of prolonged tension or pressure (Rotter). When inco-ordination has developed, the variations in the muscular contractions involve varying strain on the ligaments, especially in the case of joints, as the knee, that depend for part of their support on the muscular tendons that pass by them. The knee-joint may thus become so lax that when the patient stands, the joint may become retroflexed, as in Fig. 123. No doubt also this is the result to some extent of the want of tone in the muscles, which has been called by Frenkel ***hypotonie musculaire***. Changes in the tarsal bones and articulations may cause the foot to become flat, with a projection inwards or backwards of the tarso-metatarsal articulations, and of the (often enlarged) tarsal bones. The condition has been called the "tabetic club-foot" by Charcot and Féré.*

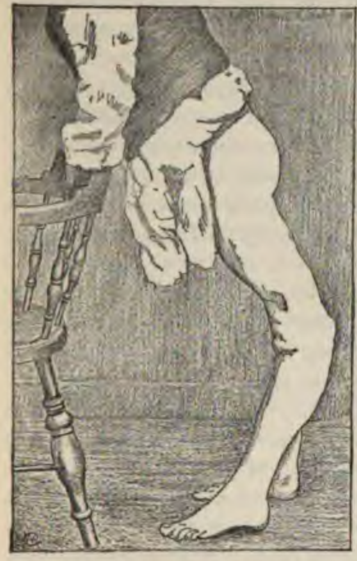

FIG. 123.—Locomotor ataxy; retroflexion of knee-joints.

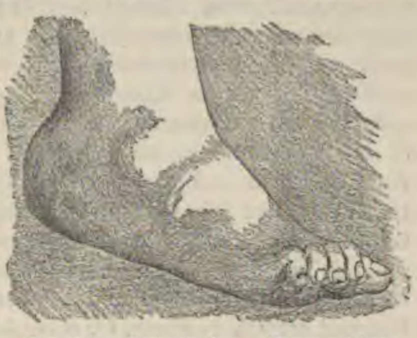

FIG. 124.—Tabetic foot. (From a photograph by Mr. Marriott, B.Sc.)

In some cases there is painless loss of teeth, and this may be accompanied by the presence of troublesome ulceration of the mouth, regarded by some as analogous to the perforating ulcer of the foot.

Visceral symptoms of peculiar character occur in many cases of tabes.† They consist almost entirely of paroxysmal disturbance of function, usually attended with great pain, and have been termed, by the French, *crises*, with qualifying adjectives according to the seat of the symptom. There is a tendency to over-elaborate this terminology. The most frequent seat of such disturbance is the stomach, and these attacks are called *gastric crises*. They consist of paroxysms of severe gastric pain, felt in the epigastrium and often passing through to the back. The pain is accompanied by vomiting, with or without nausea. The vomiting is often incessant, and is first of food, then of clear liquid, which may be very abundant; ultimately bile is vomited, and sometimes blood, which may be altered in colour; it has been ascribed to simultaneous vaso-motor disturbance in the mucous membrane. Retardation or irregularity of the action of the heart has sometimes attended such an attack, and rarely pyrexia has been noted. I have known frequent hiccough to accompany the vomiting. Such an attack lasts for some hours or days, and then subsides, to recur in a few weeks. During the intervals the functions of the stomach may be performed in a perfectly normal manner. Although pain and vomiting usually occur together, some patients have attacks of pain without vomiting, and in others there is vomiting without pain. It may occur on first rising in the morning, and be relieved by recumbency. Rarely there is nausea alone. A temperate man, in the early stage of tabes, suffered during three years from frequent attacks in which, for several days, he had intense nausea each morning, passing away in the afternoon, and succeeded in the evening by an inordinate craving for food. He never vomited unless he made himself do so, in the vain hope of thus obtaining relief. After two years, attacks of laryngeal spasm were added to the nausea. Occasionally gastric attacks coincide with paroxysms of pain in the trunk.

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* The trophic changes in bone and joint have been regarded by some as simple chronic osteo-arthritis, without causal relation to the nerve disease in their subjects, but the evidence is altogether opposed to this view. The arguments that can be adduced in support of it will be found in some of the speeches delivered at a discussion at the Clinical Society, Nov. and Dec., 1885.

† The occurrence of attacks of vomiting, sometimes early in the disease, was pointed out by Topinard, but thought by him to be a mere complication. (Topinard, 'De l'ataxie locomotrice,' Paris, 1864, p. 273.)

The chief *intestinal* disturbance is constipation, but paroxysmal diarrhœa has been supposed to be of vaso-motor origin, and connected with the disease (Pierret). Paroxysms of rectal pain (*rectal crises*) may occur, and may be accompanied by distressing tenesmus, sometimes by a sensation as if there were a foreign body in the rectum. In rare cases paroxysms of pain have precisely the character of attacks of renal colic (*nephralgic crises*), or are felt in the neck of the bladder or along the urethra or at the meatus (*vesical* or *urethral crises*). These may be attended by an intense desire to micturate, although the bladder may be empty and only a few drops of urine may be expelled. In one patient such pain was repeated several times an hour, and each attack lasted for several days. Blood may be passed after an attack.

Laryngeal crises are, perhaps, after those of the stomach, the most common. They vary much in character. The most common form is a true laryngeal spasm, with noisy inspiration and expiration, cough, and often considerable dyspnœa. The paroxysms may resemble those of whooping-cough or of laryngismus stridulus, and seem to be due to spasm of the adductors. Pressure on the superior laryngeal nerve at its entrance into the larynx, or on the trachea, or the introduction of a sound, will sometimes induce an attack. The spasm may last for a quarter of an hour or for some hours, but rarely continues so long as the gastric crises. In most if not all these cases of adductor spasm there is permanent weakness of the abductors, which when considerable constitutes a very grave complication. Whenever there is a history of laryngeal spasm, the respiratory sound should be carefully observed to detect any inspiratory stridor. In one recorded case the spasm spread to the pharynx, making swallowing impossible; a violent attack extended to the muscles of respiration, and the patient died asphyxiated. Death from these attacks is, however, rare. Paroxysms of rough cough have been termed "*bronchial crises;*" in one case such attacks ceased when the patient began to suffer from gastric crises. Such paroxysmal cough, sometimes ending with a "whoop," is more frequent than definite laryngeal spasm. The definite crises are, however, often early symptoms, and they may continue for many years. I have seen a patient, still in the first stage, who gave a history of gastric crises during the preceding eighteen years. Anginous attacks have also been described. Acceleration of the pulse is common, and cardiac disease, especially aortic, by no means rare.

Comparable to these paroxysmal visceral disturbances, although very different in character and more alarming in aspect, are cerebral symptoms; but these are, fortunately, very rare. They resemble the attacks that are common in general paralysis of the insane, and consist of transient apoplectiform seizures, of transient hemiplegia, or of convulsions, general or one-sided. They may occur early in the course of the disease. The alarm they occasion is not without foundation, since death has been known to occur during an attack of apoplectic aspect. Occasionally an actual lesion occurs (probably thrombotic softening), causing persistent hemiplegia, which is probably only coincident with the chronic disease of the nervous system. Paroxysms of vertigo sometimes occur, but their nature is uncertain. They probably frequently depend on derangement of the auditory nerve or labyrinth. Cerebral symptoms may also be superadded, especially in cases which terminate as general paralysis.

Course and Termination.—It is convenient to divide the course of the disease into three stages:—(1) In which there is no alteration in gait, the common indications of the malady being the loss of the knee-jerk and the pains, often associated with loss of the light-reflex of the iris, and unsteadiness on standing with the feet together and eyes shut. (2) That in which there is distinct affection of gait, slight or considerable, but in which the patient is still able to walk, alone or with the aid of one or two sticks. (3) In which walking is possible only with the aid of another person, or it is impossible for the patient to walk or to stand. The first stage is not always present; inco-ordination may be developed simultaneously with the loss of the knee-jerk, as one of the earliest symptoms.

The course of the symptoms is extremely variable. The inco-ordination usually supervenes gradually in the course of months, but it sometimes develops very rapidly. I have known it to become extreme in the course of a fortnight from its onset, after the first stage had existed for a considerable time. The epithet "progressive," given to the malady by Duchenne, expresses a characteristic of a large proportion of the cases in which ataxy is developed, and he knew no other cases of the disease. The inco-ordination, once developed, often increases slowly or rapidly, until it reaches a moderate or a considerable degree, and the sensory loss shows a like tendency. The local onset of the ataxy seems to be sometimes excited by a peripheral influence, such as injury; in one case an injury to the left hand was followed by inco-ordination in that limb, afterwards general and characteristic. Still more common is a rapid increase in the symptoms after such a disease as influenza, or in consequence of alcoholic excess. In such cases careful inquiry will generally elicit evidence of preceding symptoms. The pains seldom correspond to the other symptoms in degree or course. They are often very severe in the early period of the disease, and may either lessen or continue unchanged as the loss of sensation develops. Their persistence, even in severe degree, does not show progress in the disease; once set up, they may continue for years without any increase in the other symptoms, and seem in this to be analogous to neuralgia, being evidence only of functional activity in the altered nerves.

The power of recognising the first stage of the disease, which we owe to Westphal's discovery of the loss of the knee-jerk, has enlarged, and in enlarging has to some extent modified, our conception of the general tendency of the disease. It is exceedingly common for the first stage to remain stationary for a long time, for ten, twenty, and even, in one case I have known, for twenty-five years, if the duration of the lightning pains may be accepted as proof of its existence. When all cases are taken together, and if the patients are subjected to careful treatment, the disease probably shows a progressive tendency in less than one half of the cases.

The other symptoms also do not always progress *pari passu*. Sensibility may fail although inco-ordination does not increase. Conversely, the ataxy may become greater, although sensation remains the same, and even when it shows a distinct improvement. To this we shall return in considering the pathology of the disease. The contrast between the course of different symptoms in some cases is very marked. If optic nerve atrophy develops, the spinal symptoms often remain stationary, and this is also true of visceral crises; I have known gastric crises to cease when inco-ordination came on. In one patient all the symptoms of tabes passed away, optic nerve atrophy ceased to advance, and even the knee-jerk returned, when the patient became insane. The atrophy of the optic nerves, although generally progressive to blindness, also may become arrested. It may remain limited to one eye, and even when bilateral and very definite, it may cease to increase.

The rate of increase in the symptoms of tabes, when this occurs, varies much. Sometimes it is so slow that the ataxy is considerable only at the end of several years. On the other hand, it may be so rapid that in a few months the patient is scarcely able to walk. More commonly the course is variable; periods in which the disease is almost stationary alternate with others in which the symptoms increase rapidly. The exacerbations may seem spontaneous, or may be distinctly excited by some prejudicial influence—a chill, a fall, or some excess, alcholic or sexual. They are sometimes very acute; a marked change may occur in the course of a few days, or even in a few hours. Thus I have known a patient to pass, in the course of twenty-four hours, from a condition in which he could walk fairly well to one in which he could scarcely stand. The symptoms suggested that local myelitis had occurred in the degenerating cord. Muscular power may lessen in such a sudden exacerbation, or the increase may be confined to the special symptom of the disease—the ataxy. The conditions thus developed may pass away again, leaving the patient on a little lower level than before, but they often persist without any considerable recovery.

There is nothing in the nature of the disease, in most cases, to produce death. The only direct effect of the malady, which has ended life, is laryngeal spasm or paralysis. Even the gastric symptoms, prostrating as they are, are never fatal. Patients who have reached the third stage have lived, bedridden, for twenty years. Death often results from intercurrent maladies, some of which are more dangerous to these sufferers than to others because they may develop painlessly, and attain a serious degree before their existence is suspected. But many patients die from indirect results of the disease. Kidney complications are the most common; they often develop insidiously, and only manifest themselves when life is in peril, as, for instance, by acute febrile symptoms, the exact nature of which may be unsuspected until the urine is examined. I have known a patient, still in the first stage, die thus after a few days' illness. Whenever obscure pyrexia is met with in diseases of the spinal cord, interference with the function of the kidneys, due to retention of residual urine, should always be suspected and looked for. Bedsores and pyæmia occasionally cause death, although far less frequently than in most other diseases of the spinal cord.

COMPLICATIONS.—Occurring, as most cases do, in syphilitic subjects, tabes is sometimes complicated by true syphilitic lesions of the brain or cord, generally the later lesions, such as gummata, or sudden palsy from the disease of a cerebral vessel.

Acute or subacute myelitis may occur during the course of the disease with characteristic symptoms. Such a complication must not

[illegible] with the simple acute exacerbation in the special
[illegible] occurs with remarkable rapidity.

[illegible] of the cord may develop as complications of
[illegible] may be diseased with the dorsal posterior
[illegible] ataxic paraplegia," but this is a distinct
[illegible] described. Another occasional complication is
[illegible] the local atrophy already mentioned (espe-
[illegible] tongue), and general atrophy, such as is met
[illegible] form. Thus a gentleman, who had had
[illegible] vascular disease, developed the first stage
[illegible] of knee-jerk, loss of the iris-reflex, and slight
[illegible] two courses of mercurial treatment at Aix-la-
[illegible] of the second, atrophy began in the muscles
[illegible] spread during two years until almost all the
[illegible] shoulders, and back became extremely wasted.
[illegible] according to the researches of Dejerine*)
[illegible] degeneration of the peripheral nerves; it
[illegible] The extensive wasting, as in the case
[illegible] probably due, as a rule, to degeneration in the
[illegible] the spinal cord.

[illegible] present the symptoms of tabes, while in the
[illegible] inco-ordination of disseminated sclerosis, and
[illegible] the two lesions has been demonstrated, but is very

[illegible] important and frequent complication of tabes is
[illegible] insane. The two diseases have many alliances.
[illegible] syphilis is the chief cause of general paralysis, as
[illegible] reflex iridoplegia is common in both diseases. The
[illegible] combined, and the symptoms of one or the
[illegible] Thus many general paralytics present sym-
[illegible] characteristic lesion is found after death. On
[illegible] of tabes may present slight symptoms of general
[illegible] slight optimism and mental weakness, which
[illegible] or may increase to a pronounced and pre-
[illegible] may be difficult to say in which category a case
[illegible] is sometimes said that the disease may com-
[illegible] may change to general paralysis, but a more
[illegible] the facts is the co-existence of the two affections,
[illegible] the symptoms of one or the other.

[illegible] of tabes outside the nervous system, besides the
[illegible] of the malady, valvular heart disease is, perhaps,
[illegible] and usually develops without any of the usual
[illegible] an age at which degeneration is unlikely. It
[illegible] that the coincidence is really one of cause, the
[illegible] being the result of a syphilitic process. Other

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* Dejerine, 'Revue de Méd.,' 1889.

complications are so rare as only to deserve notice when a causal connection can be traced or reasonably suspected. Coincident glycosuria has been ascribed to an affection of the medulla oblongata (or may be produced through the cervical spinal cord), but it is extremely rare. But sclerosis of the posterior columns has been shown by Williamson to occur in diabetes.*

PATHOLOGICAL ANATOMY.—In most cases the spinal cord presents changes visible to the naked eye. The posterior columns have a grey translucent appearance, which is due to the loss of the nerve-fibres, and to an increase of the connective tissue ("grey degeneration"). The whole of the posterior columns may be thus changed, or only parts of them, the distribution of the visible alteration being that of the histological changes revealed by the microscope and presently to be described. In the hardened cord the difference of tint in the diseased areas is even more distinct than it is in the fresh organ. The posterior columns are also smaller than normal, because the bulk of the connective tissue is less than that of the normal fibres: hence the shape of the cord is slightly changed.

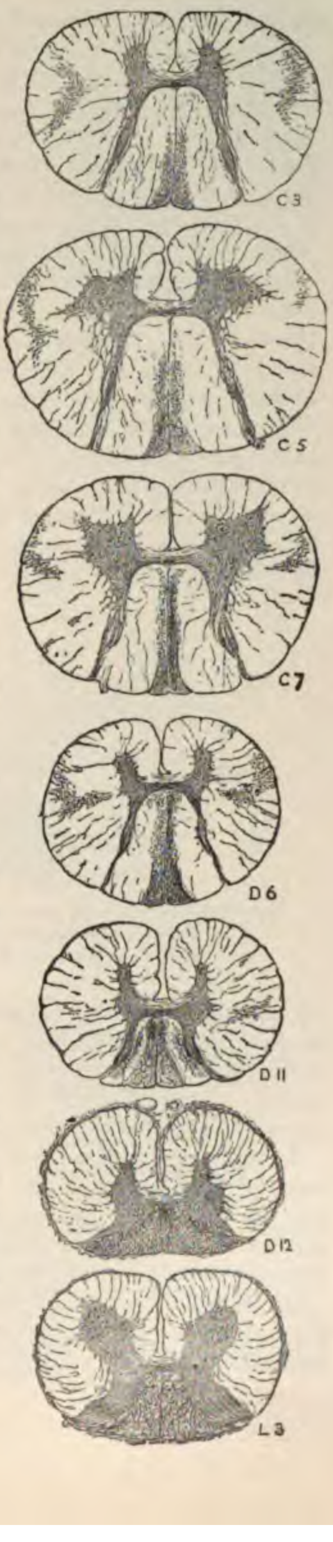

FIG. 125.—Locomotor ataxy involving the legs only. Sclerosis of the whole posterior columns in the lumbar region, gradually becoming limited, in the lower dorsal region, to the root-zone and posterior median column. The latter only is affected in the upper half of the cord (ascending degeneration). There is also conspicuous degeneration in the antero-lateral ascending tract. This is rather greater on the left side, while the post.-median degeneration is greater on the right, in harmony with the fact that the latter consists of fibres which do not decussate.

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* 'Lancet,' 1894.

In a section of the cord stained with carmine or similar reag and examined under the microscope, the affected areas are conspic by the deep staining of the connective tissue. The position of excess of this tissue, of the "sclerosis," as it is termed, indicate place in which the nerve-fibres have degenerated. In the frequent condition, in which the legs only are affected, the scle occupies the whole of the columns in the lumbar region (Fig. 125) it is often slight in the anterior parts of the postero-external colu which may even be free from sclerosis; also in the middle of columns many more nerve-fibres may be seen than elsewhere. sclerosis is most dense in the part adjacent to the posterior c through which the posterior root-fibres run, and near the surface c cord (Fig. 125, D 12). Above the lumbar enlargement the affecti the postero-external columns gradually ceases, but the degenerat intense in the postero-median columns, and has the distribution ascending degeneration, as it in fact is, receding from the commi in the upper cervical region. (Compare Fig. 125 with Fig. 77, p. In other cases the external band of sclerosis, adjacent to the c continues separate through the dorsal cord and even through cervical enlargement (Fig. 126); the median degeneration then ext

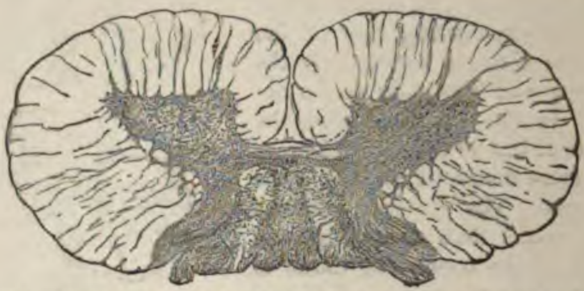

FIG. 126.—Tabes with ataxy of the arms as well as the legs. Section of cord in cervical region, showing sclerosis of the post.-med. column and root-zone of post.-ext. col. Degeneration of left ant. cornu.

up to the commissure throughout the cervical region, just as does th secondary degeneration that results from a cervical lesion (see Fig. 78 p. 216). But in the cervical enlargement, the unaffected area in th anterior part of the postero-external column is usually much large than in the lumbar enlargement.

In rare cases, of severe and long duration, the posterior columns ar occupied by connective tissue in their whole extent from one end of th cord to the other. A few fibres of the posterior roots and a fe vertical fibres near the commissure may alone be recognisable. A instance of such almost complete sclerosis is shown in Fig. 127.

On the other hand, in slight cases, in which the disease is still in th first, or in the commencement of the second stage, the sclerosis i moderate in degree, even in the regions of the cord most affected

The separation of the sclerosed areas in the postero-external and the postero-median columns may be distinct throughout the cord, except in the lumbar enlargement, where the median degeneration expands into the external column. The sclerosis of the postero-external column is then usually limited to the root-zone, and varies in width, according to that of the area through which the root-fibres pass. In the dorsal region there is usually a narrow band of sclerosis close to the posterior cornu; in the lumbar region it reaches almost to the median septum. In some instances, however, a very slight degree of sclerosis extends from the most affected tracts through the rest of the columns, and is generally greatest in their posterior half. This condition existed in the very instructive case shown in Fig. 128. The intensification of this diffuse sclerosis in the root-zone and median columns is very distinct, although the affection of the latter is unusually slight. There is, moreover, in the dorsal region, sclerosis of another part of the postero-external column—the comma-shaped tract of short fibres, which degenerates downwards for a few inches below a transverse lesion of the cord (see p. 219 and Figs. 78 and 110, F). It is most distinct in 128 at D 8, but can be traced up to the cervical region, where its form is changed.

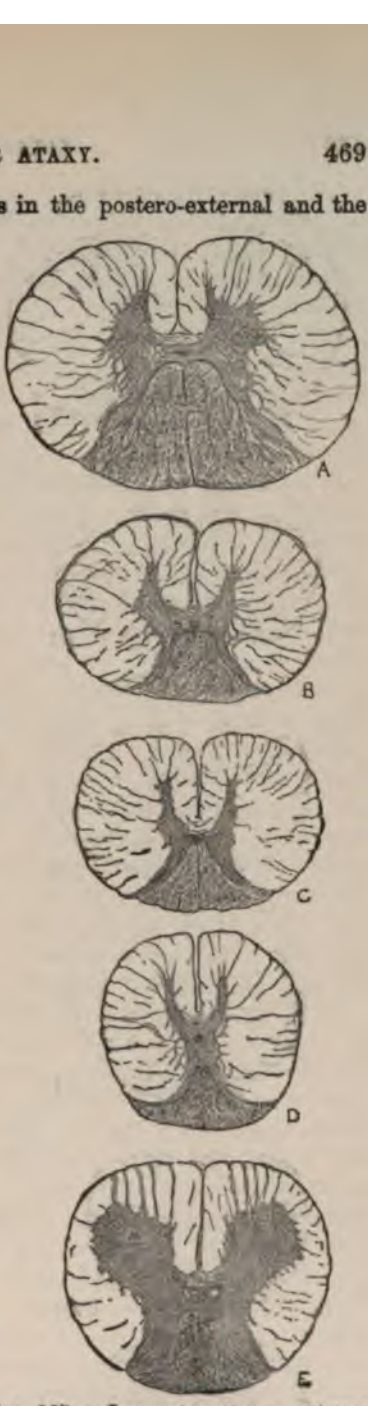

FIG. 127.—Locomotor ataxy: extreme inco-ordination and anæsthesia in both arms and legs; the posterior columns are sclerosed throughout the cord in their entire extent. A, upper cervical, B, C, D, dorsal, E, lumbar regions. In D there is also some degeneration of the intermediate grey substance.

The tract of fine fibres of the posterior root, discovered by Lissauer, is generally diseased.*

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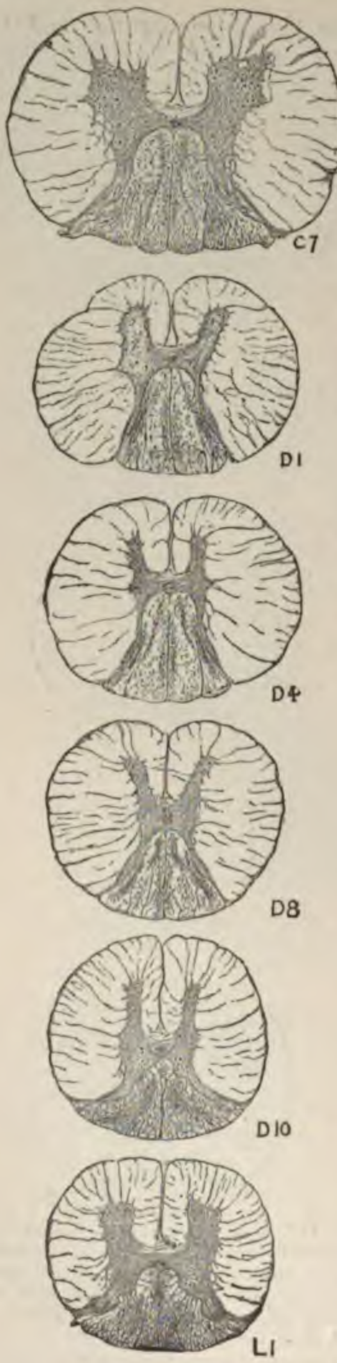
* It was first described by Lissauer in connection with its disease in tabes;



The antero-lateral columns may be perfectly normal, even when the disease of the posterior columns is great. Often, however, in old cases, there is a slight general increase of the connective tissue throughout the cord. Sometimes there is distinct sclerosis of other definite tracts, *e.g.* of the ascending antero-lateral tract (as in Fig 125), or of the direct cerebellar tract. In rare cases there is a distinct degeneration of the pyramidal tracts. The whole periphery of the antero-lateral column is thus occasionally the seat of sclerosis, the connective tissue extending in from the pia mater, which is usually also thickened; it is probable that this condition may be solely the result of the

FIG. 128.—LOCO. ATAXY; SCLEROSIS OF POSTERIOR COLUMNS.—In L 1 the posterior portion of both post.-med. col. and post.-ext. col. is densely sclerosed, but in front only the post.-med. col. At D 10 the sclerosis is also general, but is dense only in the root-zone of the post.-ext. col. At D 8 and D 4 the change is slight, except in three areas, the root-zone, the middle part of the post.-med. col., and the comma-shaped tract in the front of the post.-ext. col. At D 1 the comma-shaped tract is narrow, and in C 7 it merges in a band of sclerosis, which bounds the anterior part of the post.-med. col. There is a little diffuse sclerosis through the rest of the column, and in the first dorsal there is some degeneration of the anterior cornua.—Symptoms of the first stage had existed for four years (lightning pains, slight analgesia, loss of knee-jerk and iris-reflex, gastric crises, optic nerve atrophy). A few weeks before death the legs gradually became paralysed, and the arms ataxic; retention of urine caused acute kidney mischief, which was the immediate cause of death. No lesion was found after death to explain the subacute paralysis, but the peripheral nerves were not examined, their disease being then unknown.

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see 'Neur. Cent.,' 1885, p. 245; also ib., 1886, p. 419.

primary degeneration of the nerve-fibres even when it closely simulates the aspect of meningitic invasion. The cornu-commissural and septo-marginal tracts, the former close to the posterior commissure at the most anterior part of the posterior columns, the latter on each side of the posterior median septum, are, as a rule, spared in tabes. They probably consist of endogenous fibres, connecting different levels of the cord.*

In slight cases it is difficult to distinguish any changes in the grey matter of the cord, although it is probable that there is some atrophy of the nerve-cells and fibres in the posterior horn in many cases. Various structural changes can be discerned in more advanced cases (as Lockhart Clarke first showed), and the posterior commissure may be reduced in size. The posterior vesicular column also shows distinct degeneration in most cases. Some atrophy of the plexus of fine fibres contained in this column is said to be almost invariable (Lissauer), but frequently the cells and vertical fibres waste, and sometimes only a few shrunken cells, scattered through a translucent nucleated tissue, may remain. The atrophy of the cells is said to be especially associated with sclerosis of the direct cerebellar tract; also (but on slighter evidence) with that of large fibres in the pyramidal tract, which do not degenerate downwards. When the cells are unaffected, although the intervening substance is diseased, the cerebellar tract may be normal, and the fibres passing to it from the front of the vesicular tract may be unaffected. The degeneration of the grey matter often extends into the intermediate region between the cornua (Fig. 127, B); the intermedio-lateral tract of cells may be atrophied, and occasionally even the anterior cornu and its cells suffer in a similar way (Figs. 126, 128, D 1). As we have seen, this change may be the cause of muscular atrophy, although in other instances the muscular wasting depends on alterations in the motor nerves.

The sclerosis varies considerably in its histological characters. In slight cases there is merely a moderate thickening of the trabeculæ and their branches. The larger tracts are more fibrous than normal, and more nuclei are seen than in health. Everywhere the new growth of connective tissue proceeds chiefly from the pre-existing tracts, vessel-walls, &c. When the change is considerable the trabeculæ are very broad, and areas seem to consist wholly of connective tissue, fibrillated and nucleated. In old cases there is sometimes little appearance of fibrillation in the most affected region; cells and thickened vessels lie in a granular or homogeneous tissue. From the parts most changed the nerve-fibres have wholly disappeared; in other parts, however, close examination reveals many fibres narrower than normal, and many stained points that may possibly be axis-cylinders. The walls of the vessels are usually increased in thickness, sometimes to a very considerable degree. The coat next the endothelium may be thickened and contain many nuclei, but the chief increase, in most cases, is in the outer coat and adventitia, whence the tracts of tissue

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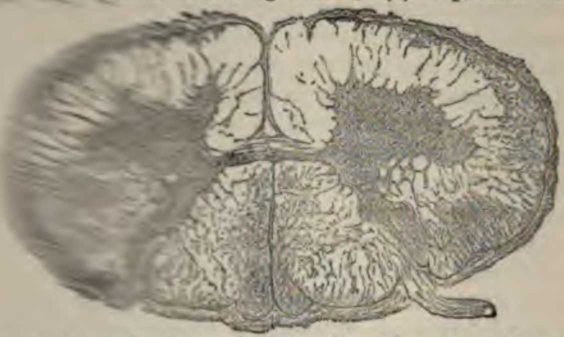
* See A. Bruce, 'Brain,' 1897.

[illegible] region. The pia mater is sometimes [illegible] columns; the vessels in it may then be [illegible] The alteration in the membrane may extend over [illegible] the lateral column, and even around the [illegible] already stated, septa passing from it into the [illegible] in the circumferential zone. This peripheral [illegible] result of three distinct processes, the relative [illegible] in different cases: (*a*) a primary degene-[illegible] ascending tracts; (*b*) a process of chronic



[illegible] Chronic meningitis. Cervical region. Sclerosis of [illegible] columns and slight diffuse sclerosis of the cord, least in [illegible] in the right lateral column. Thickened pia mater, [illegible] adjacent superficial layer of the cord.

[illegible] spreading from the pia mater, which is the seat [illegible] tissue formation; this may cause (*c*) a secondary [illegible] in the fibres affected in the first process. It [illegible] recognise this complexity of mechanism.

[illegible] course, products of degeneration, masses of myelin [illegible] corpuscles, may be found in the affected parts when these [illegible] appropriate methods.

[illegible] other lesions are found in the cord, corresponding [illegible] already described. The lateral columns may be [illegible] from any thickening of the membranes. Diffuse [illegible] met with, and, occasionally, characteristic insular [illegible] found in the cervical region.

[illegible] nerve-roots may appear normal in slight cases: it [illegible] remembered that only a considerable degree of alteration can [illegible] seen by the microscope. Often their disease is con-[illegible] when the cord is much affected they are invariably affected, [illegible] atrophied, grey, and thin to the naked eye, while the [illegible] shows wasting of the nerve-fibres and slight increase of [illegible] tissue. The intra-spinal part may be much more altered [illegible] spinal. The changes extend to the ganglia. These [illegible] sometimes normal, occasionally, however, much altered, [illegible] nerve immediately beyond them may be almost free from

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[illegible] 'Cent. f. Allg. Path. u. Path. Anat.,' 1894, Bd. v.

degeneration. The anterior nerve-roots are altogether normal, except in the rare cases in which the anterior cornua have suffered, and then some of their fibres may be degenerated, and the atrophied fibres may be traced also in the mixed nerve-trunk beyond the junction of the roots.

The peripheral spinal nerves have been found degenerated and in various degrees of atrophy in a large number of cases of tabes.* The change consists in a wasting of the nerve-fibres, beginning in the white substance, which may be reduced to a very narrow layer. Ultimately the axis-cylinders perish; they are said to present irregular swellings when the sheath begins to waste.† There may be a slight increase in the interstitial tissue and nuclei, but the change commences in the nerve-fibres themselves. The degeneration is greatest in the terminal nerve-filaments, and lessens in degree as the nerves are examined higher up, gradually ceasing in the larger trunks, and before the plexuses or corresponding main divisions are reached, the fibres are almost always healthy. The sensory fibres seem to be exclusively affected. The lesion has hitherto been found chiefly in the sensory nerves that supply the skin and joints, but that the sensory nerves of muscle undergo a similar change has been ascertained by Dejerine. It is probable that these afferent muscle nerves are invariably diseased, especially the fibres concerned in conducting the impressions that guide co-ordination, spinal and cerebellar,—on the former of which the knee-jerk depends, according to the theory maintained on an earlier page. The detection of this degeneration, among the unchanged motor fibres of the nerves, is very difficult, and this explains the paucity of evidence of it. The peripheral degeneration is found most commonly in the legs, but is met with also in the arms when these are the seat of symptoms. The change in the nerves bears no relation to that in the spinal cord in degree, or even in existence, for extreme alterations have been found in the nerves when the cord was quite normal. On the other hand, in some cases, the nerves have been healthy.

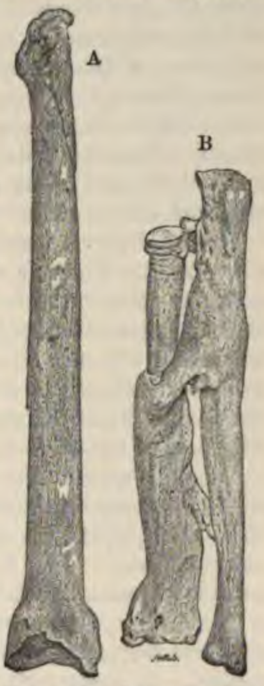

FIG. 130.—Osseous lesions in tabes. (After Charcot.) A. Atrophy of the head of the femur. B. Excessive formation of callus after so-called "spontaneous" fracture of the ulna and radius.

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* The first observation was made by Westphal, but the extent and significance of the change in the nerves was pointed out by Pierret (1880), and his observation was soon after confirmed and extended by Dejerine and Pitres.

† Schmaus, 'Deut. Arch. f. klin. Med.' 1890.

The optic nerves, when atrophied, present wasting of the nerve-fibres, and usually a remarkable increase in the interstitial connective tissue, which constitutes thick tracts of gelatinous aspect. Degeneration of the ascending or descending roots of the fifth nerve has been repeatedly found (first by Westphal in 1864) in cases in which symptoms were present in the area of its distribution. Similar changes in the roots or nuclei of other cranial nerves have also been found.

Pierret has described, as common in cases with vaso-motor and visceral symptoms, a degeneration of the intermedio-lateral tract and adjacent nerve-fibres in the upper part of the cord, traceable up the medulla oblongata in the region of the so-called "slender column," adjacent to the accessory, glosso-pharyngeal, and pneumogastric nuclei, from which it is supposed that the chief visceral and vaso-motor influences are exerted. The sympathetic has been examined in several cases, and found healthy, with one exception, in which the sympathetic on the left side of the neck was degenerated in association with left exophthalmos.

In the diseased bones or joints conspicuous changes are found after death. There is occasionally an extraordinary wasting of the articular ends of the bones, which seems to begin by rapid erosion of the cartilages, extending quickly to the bone beyond, the extremity of which may be much reduced in size and altered in form. In the hip-joint, for instance, in a case recorded by Charcot, the edges of the acetabulum had been removed, and the whole of the head of the femur and most of the trochanter had disappeared (Fig. 130).

Similar changes have been found in most of the larger joints. In the bones the compact tissue has been found thinner and more porous than normal. At the seat of old fractures a large amount of bony callus is formed (Fig. 130), and sometimes new bone is found in the capsule of joints that have been injured.

PATHOLOGY.—The great fact of the pathology of tabes is that it is a neural degeneration in the sensory nervous system, peripheral and central. In both peripheral nerves and spinal cord, the incidence of the disease is almost exclusively on the structures concerned in afferent conduction. The cerebral system suffers in far less degree than the spinal, and in more irregular manner, but in the occasional peripheral degeneration of the optic nerve, and the central changes in the roots of the fifth, we may trace the same law of distribution. It is less dominant, however, than in the spinal system, since some of the cranial nerve symptoms are exclusively motor. The motor character of the leading symptom, ataxy, is, as we shall see, only an apparent and not a real exception to the general sensory character of the malady.

The degeneration commences in the nerve-elements themselves; the overgrowth of connective tissue, which gives to the lesion its obtrusive character, is secondary. This fact has been observed in the peripheral nerves; as regards the spinal cord it rests on inference scarcely less certain. That it is true of the spinal cord has, indeed, been doubted, chiefly on the ground of the thickening of the walls of the vessels seen in some cases, and the manner in which the increase of tissue seems to start from them. But the "system" character of a disease, the limitation of a wide-spread lesion in its early stage or slight degree to structures having a common function, is probably in all cases proof of its neural origin, *i. e.* its origin in the nerve-elements themselves. In tabes the disease of the peripheral nerves depends on decay of the fibres, and the partial character of the sensory loss indicates the limitation of the chief lesion to fibres of one sensory function. There is every gradation, moreover, between the cases in which the interstitial and perivascular disease is considerable, and those in which it is so slight that there is nothing to suggest a primary interstitial process The change in the vessels may reach a high degree in the posterior median columns when their degeneration is purely secondary, and certainly commences in the nerve-fibres, being the result of a focal lesion; a similar thickening of the vessels has also been met with in secondary degenerations in other parts. It is most important to remember that the overgrowth of the neuroglia is a pathological process of growth distinguishable (and to a large extent distinct) from the degeneration of the nerve-fibres that excites it. It may vary in degree in different cases, and, even in the secondary degeneration of peripheral nerves, may assume an independent energy and even present an inflammatory character. Once excited, the process of tissue-growth may be largely independent of its cause. Secondary vascular disturbance may attend it, and thus a sub-inflammatory condition (or even a true inflammatory condition) is probably sometimes developed; it may invade the pia mater, and through this may spread widely.

The chief causal fact regarding the disease is its common relation to syphilis. Tabes is generally, although possibly not invariably, a post-syphilitic disease; certain elements of the nervous system degenerate in consequence of the influence on them of previous syphilis. There may be indeed, present and active syphilis, but, generally the active stage of the disease is over. Syphilis is often a prolonged malady, in which the results of its earlier activity sometimes coincide with its later specific effect. The elements that suffer have some special liability to degeneration, their nutrition may fail from other causes, but they seem to possess a special susceptibility to the influence of syphilis. The usual symmetry of tabetic lesions indicates that the mechanism by which syphilis affects the nervous system is a blood-state. The manner in which the peripheral nerves suffer in many cases, brings tabes, in spite of its chronicity, into analogy with the more acute forms of peripheral neuritis, such as are due to toxic agents, sometimes the result of acute specific diseases. The long interval which elapses between syphilis and tabes and the common slowness of the tabetic process are only in proportion to the chronicity of the causal malady, compared with the acute specific diseases that have neuritic sequelæ Further, the considerations mentioned in the account of multiple neuritis, which suggest that its immediate cause, when it is due to an acute blood disease, is a product of the growth of the organisms of the primary malady, rather than the organisms themselves, hold good also in the case of syphilis and tabes. The degenerative changes in the nervous system differ from the lesions of active syphilis, both in character and in time, in such a way as to make the assumption reasonable that they depend upon some product of the growth of the syphilitic organisms, a product which is probably a chemical toxine, a suggestion first made by Strümpell, and widely held regarding other analogous maladies. Recent research is bringing into ever greater prominence the important part played by such products in the generation and course of diseases. In the specific diseases that have these sequelæ, there may be more than one such product of their organisms, causing more than one kind of consequence. The differences between such nerve degenerations as those of tabes, and other late effects of syphilis, is at least not difficult to comprehend on this hypothesis; neither is the fact—which has been so great a difficulty to many—that the treatment which has so speedy an effect on syphilis itself, is generally without influence on the degenerative processes that sometimes follow it.

Tabes is probably a rare sequel, when regarded from the side of syphilis. But the occurrence of such consequences in some instances and not in others has analogies in the case of almost all the acute specific diseases that have similar sequelæ, and must be taken as evidence of variations in the precise character of the primary disease. The virus of syphilis is doubtless organismal; this has not, indeed, been demonstrated, but the analogy it presents with other specific diseases precludes doubt on the subject. Similar variations in the sequelæ are familiar to us in the case of other diseases. They sometimes have, and at other times have not, consecutive effects upon the nervous system, as in the familiar instance of diphtheria. Diphtheritic paralysis is a result of the disease, most irregular in occurrence and degree. But it deserves note that its dependence on a chemical toxin, the indirect result of the organisms, has been conclusively proved by Sidney Martin. The significance of the fact is the greater, inasmuch as its effects sometimes closely resemble the symptoms of tabes.

These considerations receive a notable emphasis from the occurrence of tabes in children who are the subjects of inherited syphilis, to whom the disease in early life is probably confined. It has been present in all the cases that have come under my notice. But the sequel differs much from those that are related to other diseases. Its occurrence is quite independent of the thoroughness of treatment of the primary disease.

The pathological facts already known enable us to understand many of the symptoms of the disease. There may be an interruption of the sensory path in one or both of two places, in the peripheral nerves, and in the posterior root-fibres as they enter the cord, and an interruption of the fibres in either place will explain the loss of sensibility which is so frequently present. The pains may most reasonably be ascribed to the molecular changes in the peripheral nerve-fibres. The intensity of a sensation is no measure of that of the process that causes it. Whether the affection of the cells of the posterior ganglia, or of the posterior cornua of the cord[*] takes part in the production of the symptoms we cannot tell. These cells are the nutritional centres of the sensory "neurons," within the influence of which are the structures that degenerate. It is noteworthy that the affection of the peripheral nerves must involve the structures by which the nerve-impulses of sensation are normally generated by mechanical and other processes. The long persistence of pains without any increase in the symptoms, shows that they may be due to the action of structures that are changed, but not changing. The loss of reflex action from the skin is explained by the interruption of the sensory path, and an increased irritability of the sensory nerves, the result of even slight degenerative changes, sufficiently explains both the hyperæsthesia and the increase of reflex action sometimes observed.

Whatever theory of the nature of the so-called "tendon-reflex" action is held, the loss of the knee-jerk must be explained by an interruption of the sensory path. On the theory I have advanced, the arrest of impressions from the afferent muscular nerves abolishes the muscle-reflex action on which the local irritability depends (see p. 25). Many other facts of disease show that this irritability is easily lost, and that a very slight change in any part of the reflex arc, too slight to cause other symptoms, is sufficient to arrest the knee-jerk.[†] Hence we can understand that this loss should be constant and early. It is not improbable, moreover, that these nerves suffer in special degree. The muscles may be insensitive to pain, *e. g.* that of electrical stimulation. Pressure and extension, which in health are painful, sometimes cause no sensation, even when the skin is sensitive.

The mechanism of the muscular inco-ordination, which is the obtrusive symptom of the disease, has been the subject of much discussion. Two fundamental facts, however, limit the problem. First, the ataxy cannot be primarily due to the loss of cutaneous sensibility.

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[*] In disease of the nerve-roots of the cauda equina there is always ascending degeneration of the posterior median columns, but not of the antero-lateral ascending tract. Hence the root-fibres from which the path is continued by this tract must end in nerve-cells in the posterior cornua, and the degeneration of this tract in tabes is proof of the degeneration of these sensory cells. See the 'Lancet,' June 19th, 1886.

[†] *E.g.* its loss after diphtheria when there are no other symptoms.

Disease of the conducting path in the cord may cause absolute anæsthesia of the skin without the least ataxy. Although this does not prove that interruption of the sensory path in the nerves, between the skin and the reflex centres, may not cause inco-ordination, this element seems to be excluded by the fact that there is no relation between the ataxy and the loss of feeling in the skin. There may be, in tabes, much ataxy without any cutaneous anæsthesia, and *vice versâ*. The second fact is that ataxy may exist in considerable degree when the lesion is solely one of the peripheral nerves, or nerve-roots, and the posterior columns of the cord are free from disease.* These two facts, taken together, seem to show that the ataxy may be produced by one mechanism, even operating alone, the disease of the afferent muscle-nerves. If the loss of the knee-jerk in tabes is to be taken, as I believe it may, as an indication of the disease of these nerves, the constancy of the loss shows the constancy of the presence of this element, in some degree. As just stated, a very slight degree of disease may abolish the knee-jerk; it is probable that a greater degree is needed to cause inco-ordination, and a still greater change to produce actual insensibility to pressure or extension.

In unilateral lesions of the spinal cord, as we have already seen (p. 272), there may be loss of the sense of posture, with intact cutaneous sensibility, on the side of the lesion, and no loss of this sense on the opposite side, on which cutaneous sensibility is lost. In such a case marked ataxy has been observed on the side on which the sense of posture was lost, when motor power returned.† Whatever effect in causing ataxy is produced by disease of the path of muscular sensibility in the spinal cord must also be produced by interruption of the path between the muscles and the cord. The latter must also arrest whatever reflex action depends upon these muscle-nerves,‡ and it is possible that such reflex action takes some part in the mechanism of muscular co-ordination.

In this connection it is important to note that the fibres that pass up by the posterior median columns seem to constitute the path from these nerves. The root-fibres to the columns pass up, as the path is proved to do, without decussating, and they may be diseased in intense degree when there is no loss of cutaneous sensibility.[*] This tract is affected in almost all cases of tabes in which there is a lesion in the cord, and in all such cases in which there is ataxy. This fact suggests that the lesion is first and chiefly of the nerve-fibres from the muscles.[†]

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* Apart from the evidence of this from cases of characteristic tabes, a very instructive case has been recorded by Dr. Hughes Bennett, in which all the symptoms of tabes were present in a case of multiple tumours of the posterior nerve-roots ('Clinical Soc. Transactions,' vol. xviii). Ataxy has also resulted from other processes, such as injury, that have caused extensive damage to the posterior roots alone.

† Gilbert; see foot-note, p. 272.

‡ In a case in which the inco-ordination was much greater in the right leg than in the left, and cutaneous sensibility was equally impaired in the two, a strong traction on the calf muscles, by forcible passive flexion of the foot, produced a distinct sensation in the extended muscles of the left leg, and no sensation in those of the right. In another case, in which the skin was sensitive to the slightest touch, the tient was unconscious of a vigorous extension of the toes produced by faradic ulation of their short extensor.

The posterior median columns (extensively connected with the cerebellum) and the direct cerebellar tract, probably conduct to the cerebellum the impressions from the sensory muscle-nerves. If so, the disease of these tracts and nerves must interfere with the co-ordinating action of the cerebellum, and cause some part of the loss of function which results from disease of the cerebellum itself.

There is another mechanism which may co-operate in increasing ataxy. The vertical fibres of the postero-external column have only a short course, and some probably connect the posterior grey matter at adjacent levels. The affection of these fibres seems to be proved by the fact that the comma-shaped bundle of fibres in the front of the postero-external column may be specially degenerated. This lesion may impair the association of the sensory structures, and so cause a want of harmony in the central mechanism. The theory that the ataxy was due to such a mechanism was advanced by Todd in 1847. It is not probable that, if effective, it has more than an intensifying influence, since ataxy may exist when the posterior columns are throughout unaffected. The discovery that ataxy may result from alcoholic peripheral neuritis confirms the view of its origin above stated.

Although cutaneous anæsthesia cannot alone produce ataxy, it may reasonably be assumed to increase that which already exists. Sensations from the skin furnish important guidance to the motor cerebral centres, and are probably also concerned in such reflex muscular actions as that of standing. Similar guidance to the cerebral co-ordinating centres is also afforded by visual impressions, the loss of which distinctly augments the defect of co-ordination. The varying characters of the ataxy in different cases probably depend on the local variations in the degree of change in the muscle-nerves. The special effect of the disease of other elements of the cord, related to the posterior roots (as the posterior vesicular column, Clarke's column), has still to be ascertained.

It is probable that co-ordination is chiefly an automatic process depending partly on muscle-reflex actions, and on the connection of neighbouring sensory structures in the spinal cord, but chiefly on the function of the cerebellum, determined by the connection of the muscles with it, and that the interruption of this connection is the chief element in the inco-ordination of locomotor ataxy. It is probable also that the automatic processes are in part under cerebral control, guided by sensory impressions which do not enter the sphere of consciousness, and that the derangement of this control will intensify inco-ordination, though incapable of producing it. We do not at present know to what extent, in any given case, the symptoms are due to the cord disease or to the peripheral nerve lesions. Apparently pains, ataxy, and anæsthesia may be due to either. The question can only be decided by the comparison of symptoms and pathological changes in a large number of cases. That anæsthesia may be due to the peripheral changes is proved by the observed correspondence of the two in distribution (Dejerine).

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[*] This posterior tract is most diseased on the side opposite to that on which there is most degeneration of the antero-lateral ascending tract.

[†] The opinion that the ataxy depends on impairment of the muscular sense was put forward in 1855 by Dr. Russell Reynolds; see p. 445. Dejerine has also associated the ataxy with the degeneration of the peripheral sensory muscle-nerves ('Archives de Physiologie,' 3rd series, vol. iii, p. 231).

The trophic changes in the skin, bones, and joints are probably due to the process of degeneration in the peripheral nerves. The degeneration has been found in all cases of the kind in which it has been looked for, and found also in the nerves of diseased joints. The pathology of the muscular wasting has been already mentioned.

The pains in the region of the fifth nerve are explained by the lesions in it root, especially in its ascending root, which, coming up from the medulla, is homologous with the posterior spinal roots. This nerve, indeed, as Pierret has pointed out, represents the sensory roots of almost all the motor cranial nerves. The degeneration of the optic nerve is fairly comparable with that of the peripheral spinal nerves. Considering the special character of the optic nerve, the absence of degeneration of the retina does not seem to destroy the analogy between the two. Regarding the pathology of the visceral crises we know but little.

The transient motor symptoms in the limbs, and in the eyeball muscles, are apparently of functional origin. They must be distinguished from the lasting palsies, which are probably due to degenerative processes in the nerves or nuclei. Such transient paralysis may precede enduring paralysis due to nuclear degeneration. The loss of function of the internal ocular muscles, being persistent, must be ascribed to degeneration of the related centres. But even the light-reflex may return (in rare cases) after it has been absent for years, and its loss must therefore depend, at least sometimes, on molecular changes capable of recovery. It is, indeed, important to remember that tabes is a disease which begins as a derangement of molecular nutrition, which is probably only to be discerned when relatively great in its degree. If we also remember that this derangement is the result of some toxic material or virus circulating in the blood, we can understand better the leading facts of the disease, and especially the variations in its course, its degree, and the precise character of the symptoms.

Diagnosis.—The diagnosis of tabes rests on the combination of symptoms already described. In the early stage, the loss of the knee-jerk, together with pains, or unsteadiness on standing with the feet together and the eyes closed, justifies a diagnosis of the commencing affection, provided we can exclude toxic peripheral neuritis (especially that due to alcohol), diabetes, and diphtheritic palsy. A lesion of the anterior cornua or nerve-roots is excluded by the absence of wasting of the muscles or change in their irritability. The diagnostic value of the loss of the knee-jerk can hardly be overrated. It is probably never absent in health. If there is doubt as to its loss, the precautions recommended on p. 21 should be adopted. When it is lost, and reflex action is in excess, a true reflex movement may sometimes simulate the jerk; the distinction is that the tap sometimes causes a movement and sometimes does not; that an interval, brief but appreciable, elapses before the movement occurs; that a similar movement is caused by a prick on the skin, and that the delayed contraction sometimes occurs in the other leg, or in the adductors of both legs.* In cases in which the knee-jerk is present, the diagnosis of tabes is only justified by distinct and characteristic inco-ordination. In the few cases of this kind that have come under my own observation, the knee-jerk has been either unequal on the two sides, or has been lost on one. Such abnormality is probably the rule in these cases, and the diagnosis is thus facilitated. In a case with lightning pains, but neither inco-ordination nor loss of knee-jerk, a suspicion of tabes would be justified by the presence of some other symptom, such as retention or incontinence of urine, loss of sexual power, or loss of the iris-reflex. As we have seen, it is probable that cases occur in which these pains exist alone,—a condition that may be termed "tabetic neuralgia."

The loss of the iris-reflex is of great diagnostic importance. It shows that a degenerative process is at work in the nervous system, and it suggests, therefore, that other symptoms are also due to degeneration. But since the iris-reflex is not always lost in tabes, the negative significance of a normal reflex is far less than is the positive significance of its loss. The practical value of this symptom can hardly be overrated. It puts the observer, so to speak, on the track of nerve degeneration. Its loss is, moreover, so often due to preceding syphilis, that it should always suggest this antecedent.

There are certain diseases with which tabes is especially liable to be confounded. One of these is multiple alcoholic neuritis. The ordinary form of this disease is readily distinguished by the symmetrical paralysis which is its chief manifestation. But the variety which closely resembles locomotor ataxy is the "alcoholic pseudo-tabes" (p. 159), in which the diagnosis may be very difficult. This is not surprising, because it resembles tabes pathologically; the lesion is a "parenchymatous neuritis," a subacute degeneration, beginning in the nerve-fibres, and similar to the peripheral form of tabes. It often affects, however, in some degree, the motor as well as the sensory nerves, and there is then some weakness in the distal portions of the limbs. An altered electrical reaction may be found in the muscles (never in the weakness sometimes met with in early tabes), and there is generally marked muscular tenderness, scarcely ever present in tabes. The pains have not the "lightning" character, and the sphincters escape. The pupils act normally, but this is not an absolute distinction, since they may be unaffected in tabes. A history of alcoholism may help the diagnosis, and so may improvement when alcohol is withdrawn. By attention to all these points, a diagnosis can generally be made without much difficulty.

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* It is impossible to exaggerate the difficulty presented by some patients in ascertaining whether the knee-jerk is or is not lost, and the care needed. Repeated observations are desirable in doubtful cases.

When the symptoms and lesion of tabes are combined with those of general paralysis of the insane, it may be doubtful in which category a case should be placed. The question is rather one of the preponderance of the symptoms of one or the other malady than of absolute distinction between them. In most cases, however, in which this combination exists, the symptoms of general paralysis become more pronounced as time goes on, and the spinal symptoms, which at first were the most conspicuous, pass into the background.

All common forms of paraplegia are distinguished by the early loss of power, and by the persistence of the knee-jerk, often emphasised by its excess and by the occurrence of a clonus in the extended rectus or calf muscles. If weakness supervenes in tabes, it is usually late in the course of the disease, and the other symptoms have been and are well marked. If the knee-jerk has been once lost in this disease, it is not reproduced by secondary lesions of the cord which ordinarily increase the knee-jerk.* The same distinctions usually suffice for the diagnosis from the combined form of paralysis and inco-ordination that I have termed "ataxic paraplegia." In this the knee-jerk is excessive, and there is foot-clonus, while pains and anæsthesia are absent; spasm supersedes ataxy. In other forms of combined disease of the cord the diagnosis depends chiefly on the recognition of union.

Acute lesions of the cord, probably situated in the postero-external column, may produce inco-ordination and pain, but the suddenness of the onset, the limitation of the symptoms, and their tendency to subside, usually suffice for the distinction. Symptoms from this cause are far more common in the arms than in the legs. Ataxy in one limb may also be produced by a tumour growing in the posterior column, but other symptoms indicate the invasion of adjacent structures, as was conspicuous in the case figured in the chapter on tumours of the cord. All these cases present the characteristics of a focal lesion with random consequences, rather than of a system disease with limitation to a special function.

The loss of perception of pain, with preserved tactile sensibility, when it involves the hands, may arouse a suspicion of syringomyelia. But the loss of the knee-jerk in tabes, and the absence of the momentary pains in syringomyelia, will enable a correct diagnosis to be made even without corroborative symptoms.

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* See, however, an exception to this, referred to on p. 455. The paper referred to mentions others.

In diphtheritic paralysis occurring long after the sore throat, or in which the nature of this was not recognised, the loss of the knee-jerk may cause tabes to be suspected; but the nature of the case is almost always rendered clear by the paralysis of accommodation and of the palate which precedes the ataxy, and should be suggested by the absence of pains and the presence of weakness. If true tabes follows diphtheria, with lasting ataxy, it is so rare as to be of little practical importance.

When the dorsal region of the cord is chiefly affected, the severity of the pains in the trunk may lead to a suspicion of disease of the vertebral bones, but in the latter the pain is local and fixed, and this, with the characteristic increase of pain when the patient moves, should prevent error. It is still more frequent to mistake such cases for neuralgia of the intercostal nerves, but even if there are no pains in the legs to suggest tabes, the knee-jerk and the light-reflex of the iris are lost in tabes. Experience shows that the most frequent errors in diagnosis are due to a mistake as to the nature of the pains, which are mistaken for rheumatism, especially when influenced by weather, or for sciatica when referred to the region of the sciatic nerve, or for some form of gout if the disease exists in the patient or his family, or in the mind of the physician.* In the rare cases in which the cranial nerves are disordered and pains are felt in the head, the symptoms may be mistaken for trigeminal neuralgia. The obscurity of the symptoms may be increased in such cases by a sense of painful constriction in the face, analogous to the girdle-pain felt around the trunk (see p. 453).

The only organic intra-cranial disease that may be taken for tabes is tumour of the cerebellum. In some cases of tabes the unsteadiness resembles that of cerebellar origin. Loss of the knee-jerk is strongly in favour of tabes; in cerebellar tumour increase of the jerk is the rule, and loss is only to be regarded as not *absolutely* incompatible. Anæsthesia and lightning pains are conclusive evidence of the spinal disorder, while in cerebellar tumour severe occipital headache is almost invariable, and considerable optic neuritis is extremely common. Optic nerve atrophy is primary in tabes, post-neuritic in tumour.

The various visceral crises are often mistaken for primary disorders of the deranged organs. Recurring attacks of the character described should always arouse suspicion of their possibly tabetic nature, and an examination of the knee-jerk will usually decide the point.

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* In one case, in which tabetic trunk-pain was chiefly felt at the level of the epigastrium, a physician of repute not only made a diagnosis of gastric gout, but refused to modify his opinion when loss of the knee-jerk was discovered, and the patient became unsteady in gait.

PROGNOSIS.—The prognosis in tabes corresponds with the facts stated as to the course of the disease. It does not, as was once thought, deserve the epithet "progressive," given to it before the pre-ataxic stage was known and before the disease had been widely observed. Arrest is frequent, and considerable improvement is not rare. The earlier the stage of the disease the better is the prognosis. In the first stage the disease often becomes stationary. When ataxy is developed the prospect of arrest is less than in the first stage, but is still considerable. Occasionally, even in the typical form of the disease, the amount of improvement is very great. One patient was scarcely able to walk across the room, in consequence of the inco-ordination, when he came under treatment, and at the end of six months his gait was scarcely distinguishable from that of a healthy person. When the third stage is reached the possibility of improvement is far less, but is not quite absent. A woman suffering from pure tabes, when admitted to hospital, had been unable even to stand for six months, on account of the extreme ataxy; but she improved so much that in a few months she was able to walk about the room without help, and has continued, now for six years, to do her household work without difficulty, and even to walk some miles with the aid of a stick. On the other hand, in some cases the malady increases in spite of every effort, and even when there is permanent arrest or improvement it is rare for all symptoms to disappear. The knee-jerk generally remains absent, and often attacks of pain continue to occur. It is difficult to find any trustworthy indications to guide the prognosis in an individual case. The fact of preceding syphilis has little influence on the prognosis. Only when the symptoms have developed rapidly and the syphilis is recent is there any possibility of benefit from antisyphilitic treatment. In all cases, however, in which the onset of symptoms is acute there is more prospect that they will pass away than if they develop slowly. In most cases the only trustworthy guide to prognosis is the observed tendency of the individual case.

Of special symptoms, the pains are often the most distressing, and unfortunately they are the most obstinate symptom of the disease. They may persist in undiminished severity when other symptoms lessen; and, on the other hand, they may become trifling when the disease increases. Diminution of the pain is a good sign if other symptoms are stationary, but not if these increase. It is often, however, a solace to the patient to know that persistence of the pains does not mean progress in the disease. The optic nerve atrophy is usually progressive, but not invariably, as is often asserted. Occasionally its progress is arrested, and more frequently when it is greater in one eye than when both are involved. Strange to say, the occurrence of optic nerve atrophy makes the prognosis as to the spinal symptoms better; in most cases the spinal lesion remains in the first stage.

TREATMENT.—The disease is one in which treatment often has a very clear influence, not only in relieving suffering, but in determining the arrest or diminution of the disease. This is true especially of treatment by drugs. But the first care should be to secure freedom, as far as possible, from all influences capable of increasing the disease, and from whatever is likely to depress the nervous system. Excessive mental work, anxiety, and physical fatigue are all harmful. In severe and acute cases, absolute rest for a week or two is often useful at the onset of treatment. If the patient walks about, exercise should always stop short of fatigue. Care should be taken to avoid the risk of falls; a severe concussion of the spine will sometimes excite to fresh activity a stationary disease. Exposure to cold is also injurious; a severe exposure may cause acute mischief. If practicable it is well that the winter should be spent in a warm and dry climate. A sea voyage is sometimes useful, combining, as it does, a maximum of fresh air with a minimum of exertion; it has, however, less actual effect than might be expected. If there is much ataxy, the risk of falls from the motion of the ship must be taken into consideration. It is the most useful in cases that are already stationary; such patients often return with a diminution in all their symptoms. When the disease is advancing, a voyage often does harm rather than good.

The digestive organs should be kept in careful order; an attack of indigestion or constipation is often attended by lightning pains, which cease when an aperient has acted. Food should therefore be light in quality, and easily digestible. The change from an active to an inactive life, which tabes often causes, may lead to the development of gout in an individual predisposed to it, either by inheritance or by his previous mode of life; and the tendency must, as far as possible, be obviated by regulation of diet and by frequent aperients. Smoking should be either avoided or limited to a small amount. Alcoholic excess is especially harmful; it sometimes excites a very severe exacerbation of the symptoms; this is not surprising, since, as we have seen, chronic alcoholism may cause a disease resembling tabes in its symptoms and pathology. Sexual excess is also most injurious. Many patients are preserved from this danger by the disease itself, but it is not always so. Excess seems to have a special influence on the optic nerve atrophy. Several very painful instances of this have come under my notice. A man in the early stage of tabes, with slight atrophy, some peripheral limitation of the fields, but little impairment of acuity of vision, started on a voyage from Australia to England. The day before he started he married, and when he reached this country he was quite blind.

The influence of drugs on the disease is unquestionable in many cases, and if they are used wisely, the more a physician sees of the disease, the more highly is he likely to esteem them. There is no specific for the malady, and in some cases the morbid tendency overpowers every influence that can be brought to bear upon it. Moreover, the influence of drugs is variable; that which seems to do much good in one case has no influence on another, and this is true also of the same case at different periods. Hence the therapeutic statements that have been made by various observers are somewhat discordant.

The first question is the influence of antisyphilitic agents. In tabes such influence is distinct only in rare cases, chiefly under the conditions mentioned in the section on prognosis. In those circumstances full doses of iodide of potassium should be given, and if the interval since the primary disease is short, mercury may be rubbed in until the gums are slightly affected. In cases of slow development, several years after primary syphilis, such treatment seldom does good, and sometimes does harm. But so widely spread is the opinion that every consequence of syphilis is amenable to treatment, that it is often well to avoid the suspicion of neglect, and to clear the therapeutic ground, by a course of iodide of potassium for five or six weeks. Very small doses of mercury, such as $\frac{1}{16}$ grain of the red iodide, may also be given for a longer time, and may often be conveniently combined with other drugs. It seems sometimes to have a tonic influence in these cases. But in such late cases, and in all the late nerve degenerations that follow syphilis, energetic mercurial treatment only does harm. Its depressing influence seems to increase the degenerative tendency, and to hasten the progress of the disease,—sometimes even to induce its extension.

Of drugs that have been recommended for tabes, those that are most useful are arsenic, iron, quinine, aluminium, nux vomica and strychnia, nitrate of silver, Calabar bean, belladonna, ergot, phosphorus. Of these arsenic is certainly that which most frequently does distinct good. Most of the cases in which I have known the greatest improvement to occur, have been taking it at the time. In several of these cases there was no other change in the conditions of life to which the improvement could be ascribed. The form in which arsenic is given matters little; ♏iij to ♏vij of the Liq. Arsenicalis may be given in a mixture, or $\frac{1}{12}$ gr. of arseniate of soda in a pill. Iodide of arsenic has no special advantage, and is not so well borne. This or any other drug must be continued for at least six weeks before an opinion of its influence can be formed. Rapid improvement can never be expected in so chronic a disease. But it is not well to continue any drug for more than about three months at a time. Most good is effected by alternating two or more; the return to one that had ceased to do good is often beneficial. In connection with the influence of arsenic, it is interesting to note the well-known effect of the drug in improving the nutrition of the skin, and the frequency with which the cutaneous nerves are degenerated in tabes. Quinine, or the extract of nux vomica, may be usefully combined with arsenic; or a small dose of mercury may also be given, ♏xv of Liq. Hyd. Bichlor. with ♏v of Liq. Arsenicalis, or $\frac{1}{24}$ gr. of the red iodide with $\frac{1}{12}$ gr. of arseniate of soda in a pill. Occasionally arsenic seems to irritate the nerve-centres, but smaller doses may then be borne, and may be useful. Nitrate of silver and phosphorus are less effective, but may sometimes be given alternately with arsenic. The chloride of aluminium is another agent that I have found of distinct service, 2—4 gr. twice or three times a day. It certainly diminishes the severity of the pains and the tendency to them.

Calabar bean has been recommended as producing temporary improvement in many chronic spinal diseases. Its influence in tabes is not great. Ergot has been esteemed highly by some, but is seldom distinctly effective. Belladonna is sometimes useful, especially when there is a tendency to incontinence of urine. Strychnia in the same cases sometimes produces marked improvement, especially combined with a small dose ($\frac{1}{200}$ gr.) of nitro-glycerine, which causes the blood containing the tonic to pass more freely to the nerve-centres.

Counter-irritation to the spine, by blisters or the actual cautery, is often useful, chiefly in the cases just mentioned, in which the disease develops, or the symptoms increase, rapidly, or in which there is spinal pain or tenderness. It is not probable that it influences the process of degeneration, but it may lessen any secondary inflammation.

Of special symptoms that require treatment, the pains are the most important. Unfortunately they are often very obstinate, and their variability renders it difficult to estimate the influence of treatment upon them. Severe paroxysms may yield only to hypodermic injections of morphia, but this should be reserved for the most intense attacks, since its use is undesirable for a recurring symptom which may continue for years. Indian hemp is often effective; gr. $\frac{1}{4}$ or $\frac{1}{2}$ should be given every three or four hours during an attack. But the most useful agents for relieving a paroxysm are antipyrine and acetanilide. One or the other of these seldom fails; should they do so, exalgin or phenacetin or migranin may succeed. Hypodermic injections of cocaine generally arrest, for several hours, pains that are superficial in character, especially if accompanied by hyperæsthesia of the skin. The injection should be of $\frac{1}{6}$ to $\frac{1}{4}$ of a grain, at the upper part of the region to which the pain is referred. Cocaine has no influence over the deeper pains. Superficial pain is also relieved by the local application of chloroform sprinkled on spongiopiline (or lint and oiled silk). Occasionally the pains are lessened by a warm bath. Counter-irritation to the spine, as by repeated sinapisms, may give some relief. These measures, however, only relieve paroxysms, and do not prevent recurrence. Chloride of aluminium is of considerable service in lessening the tendency to the occurrence of the pains. Two to four grains may be given three times a day. It is readily soluble, and may be given in combination with other drugs.

Paroxysmal visceral symptoms often yield to antipyrine or acetanilide, but when severe, morphia alone mitigates their intensity. Slight gastric crises may be relieved by simultaneous sinapisms to the epigastrium and neck. Laryngeal spasm is usually at once relieved by nitrite of amyl or nitro-glycerine; the local application of cocaine is also useful. Weakness of the bladder is sometimes lessened by strychnia or belladonna Strychnia may be given by hypodermic injection, as recommended in the chapter on muscular atrophy.

Blisters or sores on the feet should receive careful local treatment until they are well. If neglected they may become very troublesome. The patients should be cautioned never to cut a corn; a perforating ulcer is often set up by a corn being cut too deeply. The epidermis should be softened with an alkali and rubbed off with pumice-stone.

Attention to the bladder is one of the most important elements in the treatment of tabes, as in that of all diseases of the spinal cord, chronic and acute. If there is any reason to believe that the bladder is imperfectly emptied, a catheter should be passed; and if residual urine is found, the bladder should be emptied perfectly and washed out every day or every other day. No hesitation need be felt in the use of the catheter in these cases, provided very strict antiseptic precautions are taken. Many patients die every year from want of the catheter, because it is thought that all is right since they pass urine freely. The residual accumulation, or the slight cystitis, sets up chronic pyelo-nephritis, which develops insidiously, and is unsuspected until mysterious febrile disturbance occurs, chronic or acute, and leads to death. It is probable that the onset of the final acute disturbance is sometimes determined by the passage of a catheter, just as this will cause a rigor or transient hæmaturia in a healthy person. I have seen many melancholy instances of death from the consequences of unsuspected retention, while the early and frequent use of the catheter is comparatively innocuous. It is especially important to insist on the danger that the practitioner may be misled by the patient, who thinks that because he passes urine freely he necessarily empties his bladder perfectly.

Electricity has little influence on the chief symptoms of tabes. The voltaic current is powerless over either the pains or the ataxy, whether it be applied to the spine or the limbs. Faradisation of the skin by the wire brush has been recommended for the defect of cutaneous sensibility, but it has little if any influence. Temporary improvement in gait, and sometimes a little permanent improvement, may be obtained by faradisation of the muscles; the stimulation of the afferent muscle-nerves is probably the mechanism of its action, but I have not met with permanent good from its influence. When the bladder or its sphincter is weak, faradisation from the hypogastrium to the perinæum may be employed, and occasionally seems to do some good. In secondary wasting of the muscles an attempt may be made to maintain their nutrition by stimulating them with whatever current they respond to, but the central nature of the cause usually renders local treatment ineffective. Massage also has been recommended, and possibly may be beneficial in some cases. It is most useful if combined with the systematic use of the exercises in co-ordination devised by Frenkel.*

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* 'Deutsch. med. Wochenschr.,' 1896.

Nerve-stretching was fashionable a few years ago, the operation being usually performed on the sciatic nerve. In the first case in which it was employed (by Langenbuch) the procedure was followed by remarkable and mysterious improvement, not only in the pains, but also in the inco-ordination. The patient subsequently died under chloroform, administered in order that the nerves of the arms might be stretched. The spinal cord was found by Westphal to be healthy. Although the nerves were not examined, there can be little doubt that it was one of the cases in which the nerves alone are diseased. The operation has since been performed in a large number of cases, but without results that have secured its survival, and it is passing into merited disuse. Its place was taken by extension of the spinal column. In this procedure the patient is suspended in such a manner as to allow the weight of the body to rest on the head and shoulders, and head alone, alternately, and the ligaments of the spinal column are supposed to be so stretched that the spinal cord and its nerve-roots are elongated. The chief effect is produced on the ligaments of the upper part of the spine, but the actual amount of elongation of the spine is exceedingly slight and insignificant, as has been proved by experiments on the dead body. It is difficult to conceive any mechanism by which it can do the good in tabes which was at first ascribed to it; and the careful observations by Russell and Taylor on a large number of cases at the Queen Square Hospital* seem to show conclusively that it has no real power of permanently influencing the disease.

It is important that the patient should avoid falls as far as possible, and likewise exposure to cold and wet. Both of these influences occasionally excite an acute exacerbation of the pre-existing malady.

PRIMARY SPASTIC PARAPLEGIA

(PRIMARY LATERAL SCLEROSIS).

The morbid state thus designated is one that has been, and still is, the subject of much discussion. Its relations are complex, and a somewhat lengthy pathological introduction is necessary.

General Pathology.—We have seen that in every kind of transverse lesion of the spinal cord, provided this is situated above the lumbar enlargement, the paralysis of the legs is soon accompanied by excess of myotatic irritability (increased knee-jerk, foot-clonus), and that the muscle-reflex action on which this irritability and muscular tone seem to depend, gradually increases to tonic spasm, so that a condition develops to which the term "spastic paraplegia" is applied. A condition quite similar to that which is thus secondary to a transverse lesion often develops gradually, without any indication of a

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* Russell and Taylor, 'Brain,' Summer Number, 1890, vol. xiii, p. 206.

primary focal disease, and without any sensory symptoms to indicate that the mischief extends beyond the purely motor elements of the cord. We have also seen that these symptoms indicate disease of the upper segment of the motor path, the cortico-spinal segment (p. 213), which extends from the motor cortex through the pyramidal tracts, and ends in the grey matter of the cord, doubtless by a subdivision and ramification of the nerve-fibres in the fibrillary network of the spongy substance. From the gradual onset and limitation of the symptoms in these cases it has been assumed that the disease consists of a primary sclerosis of the pyramidal tracts, *i. e.* in a degeneration of the fibres of this upper segment. Since these tracts run chiefly in the lateral columns the disease has been termed "lateral sclerosis." The clinical features presented by these cases, and their probable significance, were first pointed out by Erb.* Degeneration of the pyramidal tracts had been already observed by Türck (1856) and Charcot (1865). Erb's inference as to the nature of these cases was supported by the independent (and indeed previous) researches of Charcot on cases of muscular atrophy; he showed that in such cases muscular rigidity coincides with degeneration of the pyramidal tracts. Pathologists have since been searching for confirmation of the hypothesis—for evidence that the symptoms, in their pure form, without muscular atrophy, depend on degeneration limited to the pyramidal tracts. Such degeneration in slight degree, associated with slight symptoms, has been found in cases of general paralysis of the insane (by Westphal and others); but in all other cases that have been examined either other parts of the white substance have been degenerated, or the disease has involved also the anterior cornua.† In some cases, as one published by Dreschfeld (of which a figure is given at page 497), the change in the anterior cornua has been so slight that the required conditions are nearly fulfilled.‡

Although absolute demonstration has not yet been furnished, the indirect evidence of the correctness of the pathological hypothesis is very strong, and the only open question is whether, when there is no disease of the motor nerve-cells of the anterior cornu, the degeneration is so limited to the pyramidal tracts as to constitute a system disease in the strict sense of the word. It is not surprising that demonstration of the nature of the pure cases is not forthcoming, since, as we shall see, the disease has little tendency to shorten life. It is only recently that it has been shown that the wasting in the anterior horn cells which underlies progressive muscular atrophy is

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* In 1875, 'Berlin. klin. Wochenschr.,' No. 26; 1877, 'Virchow's Archiv,' Bd. lxx.

† In a case recorded by Stofella no other part than the pyramidal tracts could be seen diseased on naked-eye observation, but no microscopical examination was made.

‡ The same is true of several cases published recently. One of Strümpell's ('Deut. Ztschr. f. Nervenheilk.,' 1894, v) very nearly fulfilled the requisite conditions.

not necessarily invariably associated with lateral sclerosis, and there seems no reason why a similar degeneration should not exclusively affect the upper segments of the motor path just as it, rarely no doubt, affects only the lower segment. In the majority of instances, of course, both upper and lower segments are affected in the condition known as amyotrophic lateral sclerosis (*q. v.*).

It should, moreover, be noted that cases which present this group of symptoms may be very various in nature. We have seen (p. 254) that, in each segment of the motor path, the same symptoms are produced by disease of any part of the segment. In the upper segment, with which we are now concerned, the symptoms are the same, when the disease is in the cortex of the brain, the internal capsule, the pyramids of the medulla, or the pyramidal tracts of the cord; and they must also be the same if the disease is limited to the termination of the segment in the grey matter of the cord. In cerebral hemiplegia the state of the arm closely resembles that present when primary spastic paraplegia involves this limb. The leg in hemiplegia presents a less close resemblance to its condition in spinal disease, because the leg is innervated from both cerebral hemispheres, and the supplementary influence of the hemisphere of the same side lessens the effect of the disease of the hemisphere of the opposite side. But if there is disease of the leg-centres in both hemispheres, the state of the legs may be identical with that resulting from disease of the spinal cord. Such bilateral disease often results from injury during birth,—meningeal hæmorrhage over the upper part of the central convolutions. This resulting condition is termed "congenital spastic paraplegia." (See Vol. II.)

On the other hand, it is extremely probable that degeneration begins in the termination of the fibres in the grey matter, just as it does in the extremities of the peripheral nerves, in each case that part of the neuron furthest from its nutritive centre. It may even be limited to these terminal parts, as the extremity of the lower segment is paralysed alone by many agents, typically curara. In such disease the white columns would be found normal, as in one recorded case in which the symptoms of spastic paraplegia existed during life, and no anatomical change was discovered after death. The detection of the disease of the terminal portion in the grey matter is extremely difficult, perhaps impossible, because the structure must consist of nerve-fibrillæ that interlace with others that are unaffected. This view is supported by the fact that in other cases some degeneration has been found in the lateral pyramidal tracts in the lumbar region slighter than the intensity of the symptoms suggested, and gradually lessening, to cease higher up the cord. Such a condition is comparable to the degeneration of the lower portion of the second segment of the motor tract in some cases of peripheral (degenerative) neuritis, in which there is a degeneration of motor nerve-fibres, greatest in their extremities, extending for a variable distance up the nerves, but lessening long before the spine is reached. It is highly probable that the toxic agents which cause lathyrism and pellagra (*q. v.*) act on the extremities of the pyramidal fibres as curara does on the nerves; among the spinal symptoms of lathyrism are paralysis of the legs with rigidity and contractures, and increase of myotatic irritability—foot-clonus, &c.*

Both clinical and pathological evidence shows that the morbid state often occurs also as part of a more extensive degeneration. Degeneration may occur in both lateral and posterior columns, giving rise to combined paralysis and ataxy—"ataxic paraplegia," sufficiently definite in its clinical characters and course to make its distinction convenient; it is therefore separately described. The following account of the clinical features of spastic paraplegia is founded on cases in which the motor paralysis and spasm existed alone, with no definite sensory loss, in which they came on gradually, with nothing in the state of the patient or the history of his symptoms to suggest either a focal lesion or an acute process.

CAUSES.—An inherited neuropathic tendency is to be traced occasionally in this as in other chronic spinal diseases. Thus in one case there was a history of insanity in uncle, aunt, and two cousins. The disease affects both sexes in almost equal frequency, presenting in this a contrast to posterior sclerosis. The period of life at which it most frequently begins is between twenty and forty; about three quarters of the cases begin in these two decades, and about an equal number in each. Cases frequently commence, however, in the second decade of life, chiefly in its latter portion. After forty they become much less common; the latest age at which I have known a typical case to commence is sixty-one.

The disease sometimes follows syphilis in a way to suggest a causal relation, even when all cases are excluded in which there is any reason to suspect a focal lesion. In one case the symptoms commenced six months after the primary disease.† But this antecedent is not frequent, and in this respect also the disease contrasts with posterior sclerosis. Proximate causes are to be traced only in a minority of the cases. The most frequent is concussion of the spine, such as a fall on the back. Some time, often two or three years, elapses between the fall and the first pronounced symptoms of the disease, and hence focal lesions due to the fall can be excluded. Next in frequency is repeated exposure to wet cold. Very rarely the symptoms have slowly followed some acute illness. In several cases the disease has succeeded prostration after childbirth or abortion, or has commenced during lactation. In one case an attack of arthritis in the knee and ankle of one leg was the immediate antecedent, and this leg was the first to become weak. In another case the symptoms followed subacute arthritis of both knee-joints, apparently rheumatic in nature.

* See Marie, 'Prog. Méd.,' 1883, No. 43.

† The cord lesion was found to be sclerosis, but the direct cerebellar tract was also degenerated (Minkowski).

The possibility that a joint inflammation may be of spinal origin must be borne in mind in considering the significance of such cases, but it is on the whole probable that a primary joint affection is an occasional cause of the spinal disease.* (Acute arthritis, certainly of spinal origin, has only been observed in severe myelitis.) The cause of the congenital form is always injury to the brain during birth, in most cases meningeal hæmorrhage, causing compression of the motor cortex.

SYMPTOMS.—Weakness of the legs, of very gradual development, is the first symptom. The patient finds that he gets tired more readily than before, and that the legs feel heavy; sometimes one leg becomes weak before the other. The progress of the weakness is very variable, but it is slow in all characteristic cases. In many instances the patient is still able to walk a mile or two, even after the disease has lasted for several years, slowly increasing. On the other hand, walking power may be almost lost at the end of six months. It is doubtful whether more acute cases belong to this category. The early weakness is often accompanied by slight unsteadiness, chiefly subjective. When the patient seeks advice, it will generally be found that there is very distinct loss of power in the flexors, often greatest in the flexors of the hip, but considerable also in those of the knee and ankle, and occasionally much greater in the last than elsewhere. The knee-jerk is excessive and quick; the rectus contraction can be obtained, as the patient lies, by tapping the depressed patella, and a rectus-clonus is often obtainable by sudden depression of the patella. The foot-clonus is usually also obtained with readiness. In rare cases, in which the upper part of the legs suffers most, there may be a rectus-clonus, but only slight indications of a foot-clonus, two or three movements quickly ceasing. The tendency to spasm is at first noticeable as slight stiffness of the legs on first rising in the morning, but it gradually increases in degree as power lessens, until at last the legs, whenever extended, pass into a condition of strong extensor spasm, rigidly fixing them to the pelvis, so that, as the patient lies, if one leg is lifted from the couch by the observer, the other leg is moved also. The spasm may be such that the knee cannot be passively flexed by any force that can be applied to it until the spasm has become less. When flexed the limb is comparatively supple; but if it is then extended, the spasm instantly returns, making the limb rigid, and often completing the extension, just as the blade of a knife opens out under the influence of its spring, "clasp-knife rigidity." The spasm is, roughly speaking, proportioned to the loss of power, and its extensor character may enable the patient to stand, the legs being fixed and rigid, when muscular power is quite insufficient to support the body when it is unaided by the spasm. In a still greater degree a voluntary effort may only excite general spasm of the limb instead of causing a definite movement. Occasionally there are also paroxysms of brief flexor spasm causing the legs to draw up. This occurs chiefly when the patient is in bed, and especially during sleep. When the spasm is great, a paroxysm may pass into violent clonic spasm, but this is merely the same clonus that can be produced by passive extension, excited by the tension of the spontaneous spasm (see p. 263). Reflex action from the skin is also usually excessive, and the stimulus excites an attack of spasm.

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* See Arthritic Muscular Atrophy.

The gait of the sufferers from spastic paraplegia is very characteristic. The legs seem to drag behind the patient, and, in walking, each is hauled forwards as a rigid whole, the toes catching against the ground, and, when the ball of the foot rests on the ground, the limb may shake from the clonus developed by the extension of the calf muscles. When the patient sits, a similar trepidation occurs until the patient pushes the leg forwards so that the heel rests also on the floor. The muscles of the legs are usually large and well nourished. They often seem, indeed, to be hypertrophied, and perhaps are really so; the contraction of spasm constitutes a physiological stimulus to growth, just as does voluntary effort. But they are not always large; they are sometimes moderately wasted. The electric irritability is usually perfectly normal, but in the muscles that present the slight wasting just mentioned there may be a trifling diminution of irritability to faradism and voltaism alike. When the patient ceases, or almost ceases to walk, contracture sometimes occurs in the calf muscles. An active contracture is indeed common, preventing at first passive flexion of the ankle beyond a right angle; but if the pressure is maintained, the muscles yield and full flexion is possible. But in some cases there is the fixed contracture described on p. 257 (form 3). Flexor contraction at the hip or knee may occur also in the same cases under the influence of posture, but is on the whole rare, the tendency to it being counteracted by the extensor spasm.

The arms are often unaffected, but they suffer in some cases, and present the same progressive weakness and excess of myotatic irritability. Vigorous contractions are elicited by a tap on the tendon of a muscle, or on the bone to which the tendon is attached, provided the muscle is extended. A clonus can sometimes be obtained in the flexors of the fingers. There is less paroxysmal spasm than in the leg, but much rigidity of the limb from active muscular contracture, flexing the elbow and flexing the fingers at all the joints, as in the "late rigidity" of hemiplegia. The difference in the form of spasm in the arm and leg is no doubt connected with existence of a reflex extensor mechanism, in the lumbar centres, concerned in the act of standing. The muscles of the arm are often as well nourished as those of the leg. The affection of the two arms is very rarely equal; it is common for one arm to be almost normal when the other is much affected and both legs are equally involved. Sometimes, however, the arm and leg on one side are paralysed, and the limbs on the other side are so slightly involved that, in comparison with the palsied members, they seem normal; the patient often believes that they are unaffected, but examination always reveals some weakness and an abnormal degree of myotatic irritability.

The muscles of the trunk may also suffer. A subjective "feeling of weakness" in the back is often complained of early, but seems to be as much a sensory as a motor symptom. Painful spasm of the abdominal muscles may occur in severe cases, and I have even known attacks of tetanoid rigidity of the back to be produced by attempts to move.

It is not common, in typical cases, for the symptoms to extend into the region of the cranial nerves, but, in rare instances, difficulty of swallowing and of articulation has existed, due to a similar palsy of the bulbar nerves. Excessive myotatic irritability has also been observed in the muscles of mastication, so that a tap on the chin causes a vigorous elevation of the depressed jaw (Beevor).

Sensory symptoms are often entirely absent, with the exception of slight dull pains in the legs, or more frequently in the back. The sensation of weakness in the back, already mentioned, occasionally reaches an intense degree. Rheumatoid pains may be troublesome, but sharp pain is rare, except in untypical cases to be presently mentioned. In such cases also there may be very slight defect of sensibility—such, for instance, as blunting of tactile sensibility on the finger-tips. Subjective sensations of "numbness," tingling, formication, are more common. They may exist for years without the development of any other anæsthesia, in spite of the progress of the motor symptoms.

The sphincters are sometimes affected, occasionally very early; often, however, they escape even to an advanced stage. Sexual power may be lost, or may remain, even when the spastic paraplegia has reached a high degree. The nutrition of the skin and joints undergoes no change.

Ocular symptoms are rare, with the exception of nystagmus, which will often be found if searched for. The action of the pupils is usually perfect, and I have only once seen optic nerve atrophy in an uncomplicated case.

The infantile form may resemble very closely that which occurs in adults. There are the same extensor spasm and increase of all forms of reflex action. As the child sits on the knee or a chair any sensory stimulus will make the legs shoot out in spasm (Fig. 131). But the rigidity does not reach the extreme degree often attained in the common form. The excess of the knee-jerk is always distinct, but a foot-clonus is not often to be obtained, perhaps because the muscle-reflex mechanism related to the calf muscles has not received the functional development that must result from the process of walking, with its recurring sequence of tension and contraction. The active contracture in the calf muscles, which most cases present, is a serious hindrance to walking even when the muscular power is sufficient, and it is long [illegible] contracture. In most cases, however, [illegible] the power of walking, although much [illegible] some peculiarity of gait, sometimes [illegible] progression," in which one foot gets over [illegible], or with a swinging oscillation of

[illegible]

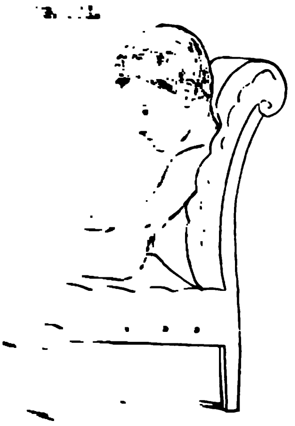
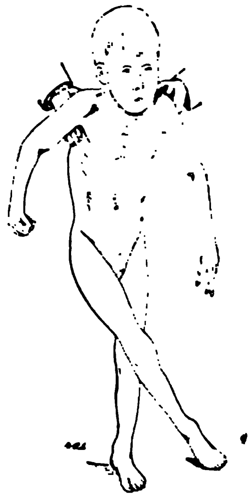


Fig. 132.



[illegible] paralysis (cerebral). Extensor spasm in [illegible] depression. (Drawn by Dr. H. R. Spencer, [illegible] Marriott.)

[illegible] paralysis of cerebral origin: cross-legged [illegible] Spencer, from a photograph.)

[illegible] to adult life. The growth of the legs is

[illegible] tonic spasm such as is seen in adult cases. [illegible] disorder of movement, spontaneous irregular [illegible], but in the cases that can fairly be [illegible] the arm symptoms are slight. When [illegible] is often termed "double athetosis." Its [illegible] Vol. II.

[illegible] met with which constitute gradations between [illegible] and other degenerations of the spinal cord. [illegible] co-exist in cases approximating the "ataxic [illegible] the next section. Cases may begin as pure [illegible] lateral sclerosis may be superadded. Slight [illegible] arms may be associated with indications of [illegible] and constitute a transition to the form of [illegible] to which the name "amyotrophic lateral [illegible]. Very rarely considerable muscular [illegible] the same part. Spastic paraplegia also [illegible] cases which afterwards develop other [illegible] disseminated sclerosis. Nystagmus, however, as has already been stated, may occur in cases otherwise offering no symptoms except those of lateral column affection. Unequivocal symptoms of syringomyelia have also been known to develop in a case which presented in its earlier stages the characteristic signs of primary lateral sclerosis.*

Lateral sclerosis of the spinal cord (like posterior sclerosis) often forms part of the morbid process that underlies general paralysis of the insane. In some cases of this character the mental symptoms are extremely slight, and the case may have the aspect of a primary spinal disease, with slight mental failure complicating it.

The course of characteristic cases of primary spastic paraplegia is thus very chronic. The malady may, at any stage, cease to advance. Slight symptoms may remain stationary for twenty years. Often, however, arrest only occurs when the disease has reached a considerable degree. It is perhaps the least dangerous to life of any chronic spinal disease. Even secondary kidney trouble scarcely ever occurs; perhaps the excessive reflex action may save the bladder from injurious over-distension. It is when other elements of the cord suffer that dangerous complications ensue.

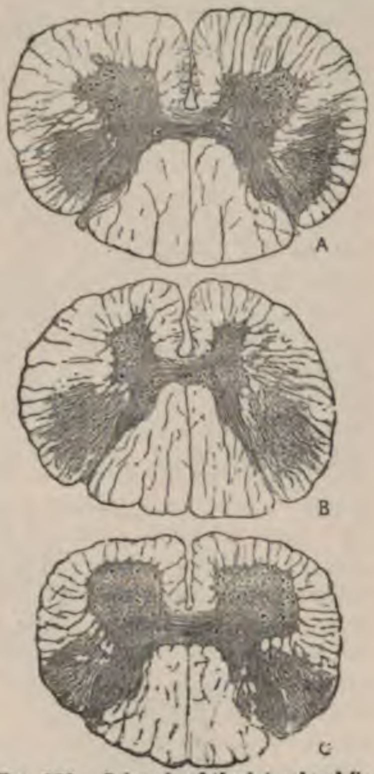


FIG. 133.—Sclerosis of the lateral and (in the cervical region anterior) pyramidal tracts, with slight degeneration of the anterior cornua. A, cervical; B, dorsal; C, lumbar sections.†

PATHOLOGICAL ANATOMY.—In the fact just stated we probably have an explanation of the silence of morbid anatomy on the subject of uncomplicated lateral sclerosis. "Nec silet mors," the apt motto of the Pathological Society, is true chiefly of disease that kills. Complete degeneration of the pyramidal tracts, anterior as well as lateral, is met with chiefly in cases in which the anterior ganglion-cells and motor nerves are also diseased, although in some instances the amount of this disease is small, and limited to the cervical region. An instance

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* Charcot, 'Progrès Médical,' 1891.

† I am indebted for these sections to Dr. Dreschfeld, who has published the case ('British Med. Journal,' Jan. 29th, 1881).

before the attempt [illegible] The degener
ever, the child [illegible] to the lateral tr
later than normal, [illegible] the cord, as in Fig.
a tendency to "cr[illegible] in the dorsal a
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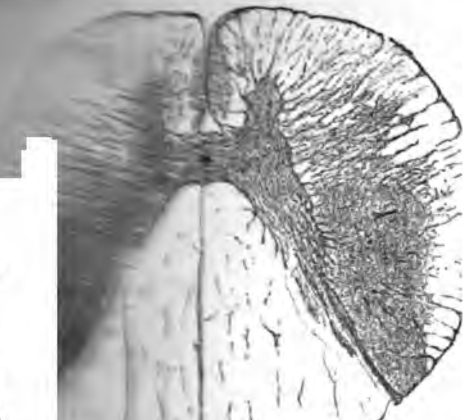

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[illegible] 'Arch. de Neurologie,' 1883, No. 18.

wards, probably in the short fibres that connect the anterior cornu at different levels. In other cases, in which the anterior ganglion-cells were normal, the sclerosis has not been limited to the pyramidal tracts; the anterior columns have been also sclerosed in the lumbar region of the cord.* The direct cerebellar tract and posterior vesicular columns have been also found degenerated with the pyramidal tract, and an annular sclerosis of the periphery of the antero-lateral column has been found in several cases.† More frequent still is a combination with degeneration of the posterior columns, described more fully in the next section. In most of these combined cases the degeneration has lessened in the upper part of the cord. In one case, at least, the white columns of the cord have been found healthy. The probable significance of this fact has been already stated (p. 491). It should be remarked, however, that there seems sometimes to be a tendency for the morbid process to extend beyond the pyramidal tracts in a manner that suggests a greater tendency for the secondary connective-tissue changes to take on an independent invasive activity than in other system diseases. (See also Ataxic Paraplegia.) Another point that deserves mention is the occasional occurrence of lateral sclerosis in association with insular sclerosis. In some cases of this character the degeneration of the pyramidal tracts is purely secondary, the result of the damage to the pyramidal fibres by an islet of sclerosis situated in some part of their course. But it does not appear that this explanation can be given of all cases. In some instances the sclerosis of the pyramidal tract appears to be independent and coincident. A similar association of insular and posterior sclerosis has also been observed; and such combinations receive additional emphasis from the fact that insular sclerosis may be accompanied by a primary atrophy of the optic nerve, similar to what occurs in tabes, although probably not identical. These facts make it probable that lateral sclerosis, although a system disease, is often one of less strict features than tabes, having some tendency towards chronic myelitis. We shall see that this is true also of ataxic paraplegia.

The probable mechanism of the symptoms that give the dominant characters to the malady have been considered in the general account of the symptoms of disease of the spinal cord. The most important fact is that the degeneration, whatever its upward extent, always involves the lowest part of the pyramidal segment, because it seems to begin in this and ascend the fibres. Hence the intra-cornual termination must always be involved. This, it is assumed, is the structure that

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* As in a case recorded as one of spastic paraplegia by Hopkins ('Brain,' Oct., 1883), but this was an example of combined lateral and posterior sclerosis (ataxic paraplegia). An illustration of the changes in this case is given in the next section.

† Direct cerebellar and pyramidal tracts in a case by Minkowski, in which the disease rapidly succeeded syphilis; annular sclerosis by Westphal (see Ataxic Paraplegia).

controls the muscle-reflex centre, and the consequent loss of control explains the excess of myotatic irritability and the progressive spasm, progressive by what may be termed a functional hypertrophy resulting from continuous over-action. When there is the rare consecutive wasting of muscles we must conceive that the degeneration involves also the ganglion-cells and lower segment of the motor path; when there is coincident wasting of other muscles than those that are the seat of spasm, it would seem that some elements of this lower segment are the seat of a primary degeneration. The slighter muscular wasting without, or with only trifling, change in electrical irritability, is the expression of a slighter alteration in the nutrition of the cells and fibres, without actual destructive degeneration.

DIAGNOSIS.—The diagnosis rests on the combination of weakness, excess of myotatic irritability, and spasm—on the gradual onset of these symptoms—and also on the absence of indications of a focal lesion. An acute onset, occupying a few days or weeks, is *prima facie* evidence of a lesion that takes the case out of the category of degenerative disease. In most acute cases there is other evidence of a focal lesion, extending, at some level, beyond the limits of the motor path. Such indications are initial impairment of sensation, or a girdle-pain. The latter indicates irritation of the posterior root-fibres at a certain level, and proves that at that level the disease extends beyond the limits of the pyramidal tracts. Spastic paraplegia is common after such lesions, but is secondary and not primary.

Although marked sensory symptoms, in a case that presents the symptoms and course of primary lateral sclerosis, are thus evidence that the sclerosis extends beyond the motor tracts, and that the case is not one of pure lateral sclerosis, it is doubtful what significance is to be attached to very slight sensory symptoms, such as slight subjective sensations of dull pain, formication, &c., when they exist alone. It is possible that they are due to functional disturbance in the sensory nerve-elements, and do not imply structural disease outside the motor area. They are not followed by any more pronounced sensory symptoms.

The absence of objective unsteadiness is the chief distinction from ataxic paraplegia (*q. v.*). When this is slight it may be difficult to say in which class a case should be placed. There are no doubt intermediate cases in which there is a very slight degree of the additional lesion that exists in ataxic paraplegia, but the distinct objective character of the ataxy is the best criterion. Considerable muscular wasting in any part is commonly regarded as bringing the case into another category, that of "amyotrophic lateral sclerosis"; but some cases of the kind, in which the wasting is confined to a few muscles, resemble more closely the cases we are now considering than they do those with wide-spread and extreme muscular atrophy. These cases are further considered in the account of progressive muscular atrophy.

Primary spastic paralysis is not uncommon in those who are at the age and of the sex at which hysteria prevails, and there is no form of cord disease that is so often mistaken for hysterical paraplegia. The mistake is facilitated by the perfect muscular nutrition. But the mistake ought not to occur, as the peculiar extensor character of the spasm, lessening with flexion, is distinctive; nothing resembling it ever occurs in hysterical paraplegia. When the spasm is trifling or absent, as in slight and early cases, the diagnostic difficulty is much greater, and is increased by the fact that slight excess of myotatic irritability occurs in some cases of so-called hysterical paralysis. But this scarcely ever reaches the degree necessary to give rise to a true foot-clonus or a rectus-clonus. There may be a slight clonus produced through a voluntary depression of the foot of the patient in response to the passive flexion of the ankle, and readily recognised; or a true clonus may be obtainable during hysterical contracture, but apart from such contracture a true foot-clonus or a rectus-clonus deserves the greatest weight, as all but conclusive evidence of organic disease. I have known many mistakes in diagnosis, in which lateral sclerosis was mistaken for hysterical paraplegia owing to disregard of the evidence afforded by this symptom, but I have never known the opposite error from undue regard to this symptom. Moreover an excess of myotatic irritability in so-called hysterical paralysis must depend on more than functional disease. There must be changes in nutrition, and consequent persistent defective control of the muscle-reflex centres. On the hypothesis that I have advanced, this control is exerted by the termination of the pyramidal fibres, *i. e.* of the upper motor segment, by the structures degeneration of which probably causes spastic paraplegia. A case is actually on record (the case of lateral sclerosis described by Charcot in 1865) in which an initial hysterical paraplegia, cured suddenly and relapsing on emotion, passed ultimately into lateral sclerosis. Similar cases of initial hysterical paraplegia frequently develop all the characters of disseminated sclerosis.

When the arm and leg suffer on one side only, the disease may be mistaken for cerebral hemiplegia. There is not, however, any affection of the face, which, although theoretically conceivable, is, as a matter of fact, always absent. The limbs on the other side are never quite normal, but present slight symptoms—weakness and an excess of myotatic irritability—similar to those on the affected side, which indicate the nature of the malady.

The diagnosis of the congenital infantile form is only difficult when the observer is unaware of the occurrence of these cases. A slight degree of inco-ordination in the hands will usually be found if they are carefully watched while the patient takes hold of some object. The wide separation and irregular movement of the fingers is very characteristic. Chronic primary cord diseases are almost unknown in young children. Caries of the spine is, in them, the chief cause of paraplegia; and the definite onset of the paralysis, in a previously healthy child, is an absolute distinction from the cases of birth-palsy. Other diagnostic indications are described in Vol. II (Infantile Meningeal Hæmorrhage). A definite onset also distinguishes glioma of the pons, which may likewise cause spastic paralysis of arms and legs.

PROGNOSIS.—In primary lateral sclerosis there is some prospect of arrest, and even of improvement, if the disease has not reached an advanced stage. Actual recovery is rare, but does sometimes occur in early cases. When the spastic state is well developed and has lasted for some time it is very rare to obtain more than arrest. This is, perhaps, due rather to the tendency of the disease than to its degree, since a similar condition that is secondary to a focal lesion often passes away completely; and many facts show that it is not likely that there is anything in the nature of the lesion to preclude the restoration of a normal condition until dense connective tissue has replaced the nerve-elements. In the cases that improve most, it is possible that the disease is limited to the terminal structures. We have not, at present, any guide to the prognosis in an individual case, besides its duration and degree, except its observed tendency, especially under treatment. The prognosis of the infantile form is considered in Vol. II.

TREATMENT.—So far as drugs are concerned, the treatment is, to a large extent, the same as that of posterior sclerosis, already described. The drugs most useful are the same, but, unfortunately, their influence is less frequently appreciable. Nux vomica and strychnine have, however, to be given with caution, and in very minute doses, as they have a tendency to increase the spasm. In severe cases this is a most distressing symptom, and often not amenable to any influence. Bromide sometimes lessens it slightly, but even large doses of bromide have but a trifling effect. Indian hemp, belladonna, and Calabar bean may be tried, but seldom have a distinct influence. One of them may be combined with arsenic, or whatever metallic agent is employed. Absolute rest is sometimes of service, and occasionally seems to produce improvement, which all treatment failed to effect while the patient was walking about. The avoidance of fatiguing exertion is a very important element in treatment, and often seems to permit medicines to do good, which before were counteracted. Rubbing is also beneficial in some cases. Its influence on the spasm is often very distinct during the process, and a long course of rubbing has produced a permanent improvement in the spastic condition. Upward rubbing seems to have more influence than kneading the muscles. If there is contracture of the calf-muscles, the foot should be pressed up while these muscles are rubbed. When there are facilities for it, the rubbing may advantageously be combined with sweating in the Turkish bath. In one case, of moderate degree, almost all the symptoms passed away after a long course of Turkish baths, arsenic being also given. The patient, who could at first walk scarcely half a mile, became able to walk several miles without fatigue, and the improvement was permanent. Electricity is useless in the pure disease. Faradism and all painful applications are harmful, stimulating further the already excessive excitability of the reflex centres. The constant current to the muscles, or from the spine to the muscles or to the feet in water, has no distinct influence in either lessening the spasm or improving the strength. The actual cautery is in many cases most beneficial.

In the infantile form, drugs are useless. Rubbing is desirable, and can be efficiently performed by the nurse or mother. Carefully planned gymnastic exercises are also useful. The tendo Achillis is sometimes divided for the contracture of the calf muscles, but the operation is useless and ought never to be performed. Supports help the child to walk somewhat sooner than it would without their aid, and so hasten improvement, but they should only be employed when there is sufficient power and control to make them useful by enabling the will to effect ordered movements.

FAMILY FORM OF SPASTIC PARALYSIS.

Cases in which the usual symptoms of spastic paralysis have been present in several members of the same family, occasionally in different generations, have been described by Erb,* Strümpell,† Bernhardt,‡ Newmark,§ Raymond, Sougues,‖ Gee,¶ Tooth,** and others. Occasionally to the symptoms of the cord affection evidence of bulbar involvement has been added, and the condition has probably been dependent upon some congenital weakness of the pyramidal tracts and their correlated structures, predisposing them to premature decay. Optic atrophy has also been met with. The condition is further briefly alluded to in the chapter on Hereditary Ataxy (*q. v.*).

ATAXIC PARAPLEGIA.

(COMBINED LATERAL AND POSTERIOR SCLEROSIS.)

The term ataxic paraplegia seems the most accurate clinical designation for a disease of the spinal cord which presents a combination of the symptoms of paraplegia and ataxy, and consists in combined disease of the posterior and lateral columns. Although its clinical features present some varieties, and may approach those of each of its constituent forms of disease, yet in the majority of cases the symptoms are uniform, and sufficiently characteristic to justify the distinction of the disease and its separate description. As early as 1867 Westphal described combined sclerosis of lateral and posterior columns in general paralysis. Erb, von Leube, Prevost, Pierret, and others, during the next ten years, published cases definitely showing the simultaneous occurrence of sclerotic changes in the lateral and posterior columns. Von Leyden, in his book on spinal cord diseases in 1875, briefly referred to the condition as one of "combined sclerosis of the posterior and lateral columns," and mentioned a case which he had seen. The first systematic description of the condition, however, was that given in 1877 by Kahler and Pick in an article on "Combined System Affection of the Spinal Cord." C. Westphal next published a paper on "Combined Affection of the Tracts of the Spinal Cord," in which five cases were described. Later Dana, in America, described similar cases; Grasset, going on clinical features, described a similar condition as "ataxo-spastic tabes" and Dejerine as "ataxo-paraplegic tabes."* In the first edition of this book, published in 1886, a condition, similar both clinically and pathologically, of which the writer had seen several cases, was described under the name of ataxic paraplegia, a descriptive title indicating the combination of paraplegic weakness with ataxy.

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* 'Deut. Ztsch. f. Nervenheilk.,' Bd. vi.

† Ibid., Bd. iv.

‡ 'Virch. Archiv,' cxxvi.

§ 'Amer. Journ. Med. Sci.,' 1893; 'Med. News,' 1897.

‖ 'Sem. Méd.,' 1896.

¶ 'St. Bart.'s Hosp. Reports,' xxv.

** Ibid., xxvii.

CAUSES.—Neurotic heredity is to be traced only in a small proportion of the cases—about one tenth. A history of syphilis is as rare as it is frequent in pure tabes. Males suffer much more frequently than females. The disease usually commences between thirty and forty, but I have known it to begin as early as nineteen and as late as fifty-two, while commencement at fifteen† and at sixty-one‡ is on record. Exposure to cold is to be traced occasionally as an exciting cause. In one young lady the symptoms commenced after a season of balls, at which when heated she would habitually sit at open windows, and often sleep in clothes saturated with perspiration. In a few cases, severe exertion, such as excessive athletics, has been the apparent cause. A severe concussion of the spine has sometimes preceded the first symptoms by a few months. The disease may also follow great sexual excess. In many cases no cause can be traced, immediate or remote. Its general etiology thus resembles that of simple spastic paraplegia.

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* See Prevost, 'Arch. de Physiologie,' t. iv; Pierret, ib.; Babesieu, 'Virchow's Archiv,' Bd. lxxvi; Kahler and Pick, 'Arch. f. Psychiatrie,' Bd. viii; Westphal, ib., Bd. viii and x; Dana, 'New York Med. Record,' July 2, 1887; and Clarke, 'Brain,' 1890. As to Dejerine's description see Ladame, 'Brain,' 1890, p. 530; Grasset, 'Maladies du Systeme nerveux;' Montpellier, 1894.

† Oppenheim, 'Neur. Cent.,' 1888, p. 647. Such early cases are generally isolated forms of "Hereditary Ataxy."

‡ Suckling, 'Lancet,' 1886.

Symptoms.—The onset of the disease is usually slow and gradual, two or three years passing before walking power is much impaired; it is rarely subacute, so as to reach a considerable degree in two or three months. The early symptoms resemble those of spastic paraplegia, with the addition of ataxic unsteadiness, which may be at first the most prominent symptom. The legs suffer first, often alone, or the arms may also be involved. The patient finds that he tires more readily than before, and also becomes unsteady on turning or walking in the dark, and these symptoms gradually increase. The weakness may be slight, but if all the movements are tested, a defect in power will be found in some, often most in the flexors of the knee and hip, and frequently more in one leg than the other. The patient is unsteady when he stands with feet together, and he tends to fall if the eyes are then closed. If the feet are bare, the irregular action of the muscles is shown by the movement of the tendons on the dorsum of the foot, as in tabes. In early cases, the inco-ordination is revealed by the patient's gait, which is distinctly unsteady. There is rarely the high movement and sudden descent of the feet often seen in tabes, but (as in many cases of tabes) the patient is unsteady, reels on turning, and has often to bring his foot suddenly to the ground to maintain his equilibrium. He may even have to steady himself with a stick, or to catch hold of some adjacent object, to save himself from falling. The ataxy is equally evident when he lies, and attempts, his eyes being closed, to touch some object with his foot.

The sensory and reflex symptoms present a marked contrast to those of tabes. Lightning pains are almost always absent; I have only met with them in one case, and in this they were a transient symptom. Sometimes there is a slight dull pain in the legs, felt especially on fatigue. Dull pain in the sacral region or in the spine is not uncommon, and is often an early symptom. The sacral pain, indeed, is sufficiently frequent to deserve special note. A girdle-pain is met with only in rare cases. As a rule there is no loss of sensation either on the legs or trunk. Hyperæsthesia is equally rare. Reflex action from the sole may be normal or increased; less commonly it is diminished. The cremasteric and abdominal reflexes are sometimes lost. The most striking difference from tabes, however, is in the condition of myotatic irritability, which, in the vast majority of cases, is greatly increased. The knee-jerk is quick and extensive; it can be obtained from above (see p. 22), and there is generally a distinct rectus-clonus. The foot-clonus is also commonly to be obtained. This myotatic excess persists and increases, and when the arms are involved they present symptoms similar to those in the legs—inco-ordination, weakness, and marked excess of the myotatic irritability.

There may be conspicuous ataxy of the hands, and a tendency to cramp-like spasm on an attempt to use them. The muscles in both arms and legs are usually well nourished.

Sexual power is often lost early in the disease. The sphincters may

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Among complications the most important are mental changes resembling those of general paralysis of the insane, of which indeed this combined sclerosis may form part. Slight muscular atrophy sometimes occurs. Arthritis of doubtful significance has been observed. A patient of Westphal's presented derangement of the sympathetic, and died from peculiar spasm of the muscles of respiration. Visceral crises, however, are practically unknown.

PATHOLOGICAL ANATOMY.—In all cases, the spinal cord has presented sclerosis of both posterior and lateral columns; but the precise extent and degree of the degeneration are subject to considerable variations. As a general rule, the sclerosis of the posterior columns differs from that of tabes in two particulars. First, it is not more intense, and often it is less intense, in the lumbar than in the dorsal region of the cord. Sometimes, indeed, as in the case shown in Fig. 135, in the middle and lower parts of the lumbar region, the posterior columns may be free from sclerosis, although it is considerable in the dorsal region and at the junction of this with the lumbar enlargement (B). The second difference is that the sclerosis has not that special intensity in the root-zone of the postero-external column which characterises the lesion of tabes. In rare cases the whole posterior column in the lumbar region is diseased. The part of the external column near the commissure and near the neck of the posterior horn usually remains free. Sometimes the degeneration does not extend up to the posterior surface of the cord; for instance, as in the case figured, it affects chiefly the middle three fifths of the posterior columns. When the degeneration is considerable, the posterior median columns, in the upper part of the cord, may present the usual ascending degeneration of secondary origin. When the lesion is slight in degree in the lower half of the cord, there may be only a diffuse degeneration of the columns in the cervical region, similar to that below, and not the more intense limited affection of the median part which occurs when there is a typical ascending degeneration from a considerable lesion of the column lower down.

The degeneration in the lateral columns is also variable in extent and position, and is often not strictly "systemic" in character, *i. e.* is not strictly limited to a single system of fibres, although the pyramidal tracts are chiefly affected. In one or two cases, indeed, the whole pyramidal tract, and this alone, has been degenerated; but more often the sclerosis, while intense in this tract, also extends in front of it into the mixed zone of the lateral columns (Fig. 135, A, B). A similar extension, however, is usually met with in the sclerosis that attends degeneration of the anterior cornua; compare Fig. 133. The lateral limiting layer, between the pyramidal tract and the grey matter, may be also invaded. The direct cerebellar tract often escapes, as in Fig. 135 A (B is below its level of origin), but it is affected in some cases. Occasionally a zone of sclerosis has existed in the whole periphery of the cord, extending deeply into the lateral column in the position of the pyramidal tract. This tract has been found diseased, in some degree, in all cases, and in several the extent of its degeneration has been greater in the lower part of the cord than in the upper. The direct (anterior) tract may be affected as well as the lateral, and is commonly involved when the lesion is considerable in the upper part of the cord. As in most other degenerative diseases of the cord, there may be some increase of connective tissue in the unaffected columns. As a rule no morbid change has been recognised in the grey matter or in the membranes. In one case only was there slight meningitis. Disease of the ascending root of the fifth has been found in some cases;* in one there was also atrophy of the cells of the oculomotor nucleus, and the case thus differed considerably from the type. The muscles have been found normal.

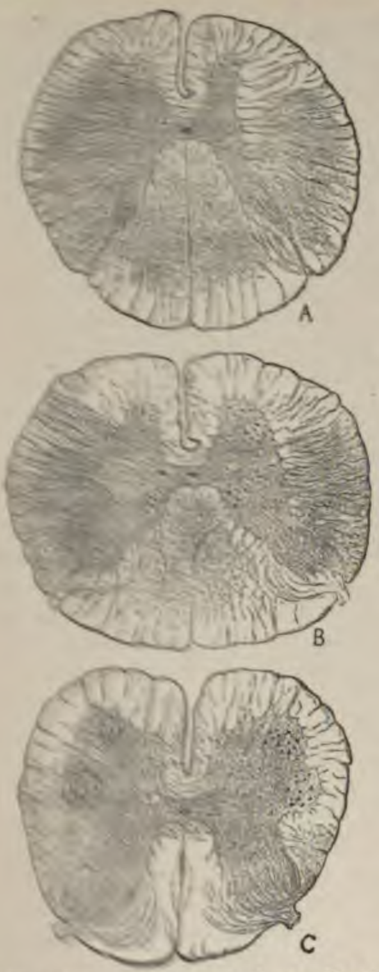

Fig. 136.—Ataxic paraplegia, combined lateral and posterior sclerosis. A, upper dorsal; B, last dorsal; C, mid-lumbar. The posterior columns are free from sclerosis in C, except in their anterior angles; in B the disease involves the middle and anterior parts of both post.-med. and post.-ext. columns except in the neighbourhood of the neck of the horn; in A the sclerosis is slighter and is confined to the middle three fifths of these columns. The sclerosis of the lateral columns in C is limited, on the right, to the pyramidal tract, on the left it extends in front of this; in B it is very dense in the whole lateral column, involving not only the pyramidal tract but the limiting layer, and part of the "mixed zone;" in A it is similar in extent but slighter in degree. There is some increase of tissue [illegible] the anterior columns, and a focus of sclerosis near the anterior fissure, on one side in A and B, on both sides in C.†

Pathology.—The double lesion in the posterior and lateral columns supplies an explanation of the two sets of symptoms which characterise the disease. The inco-ordination must be referred to the disease of the posterior columns, which, greatest in the dorsal region, involves there the fibres that conduct impulses from the muscles, probably to the cerebellum.[*] The effect must be to lessen the cerebellar guidance, and this explains the resemblance of the inco-ordination to that in cerebellar disease. The fact that the disease does not extend into the lumbar root-zone accounts for the integrity of the muscle-reflex action, and enables us to understand this marked difference from tabes. Disease of the direct cerebellar tract probably has an influence similar to that of the posterior median columns. That interruption of the conducting path in the cord will produce inco-ordination is, as we have seen (p. 258), well established. The sclerosis in ataxic paraplegia occupies also the region of the cord in which the short vertical fibres run, connecting the posterior grey matter at different levels, and it is possible that the damage to these fibres may contribute to the ataxy.

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* Oppenheim, 'Neur. Cent.,' 1888, p. 647.

† I am indebted to Mr. J. Hopkins for the opportunity of drawing these sections. The patient was a man aged twenty-one, in whom weakness of the legs commenced

The only recognisable lesion that can be regarded as the cause of the paralysis is the degeneration of the pyramidal tract. The statements made regarding spastic paraplegia apply also to the disease, and need not be repeated. The lesion has been found greater on the side on which one leg was weaker than the other. The variations in the amount of disease, which does not always correspond to the degree of palsy, are probably due to the fact that the latter may be caused by degeneration of the termination of the fibres, which may be much greater than that of the fibres higher up the cord. In most cases that have been examined, the visible disease was greater in the lower than in the upper part of the cord, increasing from above downwards, a fact which suggests that the maximum lesion is at the lowest part—in the terminal ramification within the grey matter.

The chief pathological difficulty arises from the fact, which we have already considered in connection with spastic paraplegia, that the morbid process often extends beyond the limit of the tracts chiefly diseased. It tends to assume a diffuse character, so that it has

at twenty, after a wetting, improved, and then slowly increased. He was admitted a year after the onset, with considerable weakness of the legs, a reeling, unsteady gait, increased knee-jerk, foot-clonus, but with no anæsthesia, wasting of muscles, pains, or affection of the arms. The symptoms slowly increased, paraplegia became absolute, and the spasm very intense, sometimes flexor and sometimes extensor. There was a doubtful impairment of sensibility to touch on the legs, but no loss to pain. The sphincters became affected, bedsores formed, and the patient died two years after the onset. Fuller details will be found in 'Brain,' October, 1883, p. 383.

[*] Much evidence has been obtained of the connection of the posterior median columns with the cerebellum through the grey matter of its bulbar nucleus, and also of the effect of interruption of these columns in the dorsal cord, in causing inco-ordination like that of cerebellar disease. Besides the experiments of Bechterew on the latter point already mentioned (p. 215), a recent important investigation into the connections of the cerebellum fully confirms that with the post.-pyramidal nucleus (Brosset, 'Contrib. à l'étude des Connexions du Cervelet,' Paris, 1891).

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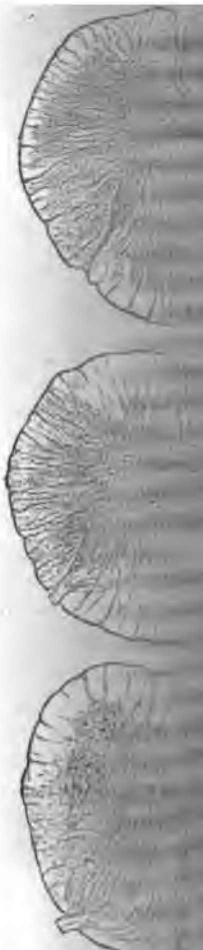

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† I am indebted [illegible] debility, &c. Some of the cases described as
The patient was a [illegible] of this nature (see next section).

the latter. The subacute forms of myelitis are also distinguished by their more rapid onset.* The greatest difficulty is presented by cases which have a subacute onset, and the distinction from a local myelitis involving the posterior and lateral columns may be very difficult. It depends chiefly on the progressive tendency of the symptoms, which contrasts with the regressive tendency of myelitis. It is possible that when there is a degenerative tendency, a focal myelitis may set up a progressive degeneration in the columns concerned, as it certainly may set up a degeneration limited to the posterior columns.

A tumour in the middle lobe of the cerebellum may cause unsteadiness, closely resembling that of some cases of ataxic paraplegia, and it may also cause weakness of the legs with increased knee-jerk, from the pressure on the pyramidal fibres as they pass through the pons. In such cases, indeed, we probably have the two elements of the disease, produced in a different manner, by lesions of different position. We have a degeneration of the lateral columns which is secondary instead of primary, and we have disease of the co-ordinating centre instead of interruption of the path to it. But the weakness in the legs is never great in cerebellar tumour, and special symptoms of this are never absent. Occipital headache, vomiting, and optic neuritis are present in most cases. In a case in which the pons is compressed, some cranial nerves are usually also damaged.

PROGNOSIS.—The prognosis in ataxic paraplegia is similar to that in the spastic form, and depends on similar considerations, which need not be here repeated.

TREATMENT.—The treatment of ataxic paraplegia is the same as that of the allied diseases, and especially of spastic paraplegia, to the account of which the reader is referred.

SCLEROSIS OF THE CORD FROM TOXIC BLOOD-STATES.

PELLAGRA.

Pellagra is an endemic malady which, though fortunately unknown in this country, deserves mention here on account of the incidence of its effects on the spinal cord. Its chief anatomical lesion is a degeneration of the lateral and posterior columns of the cord, corresponding closely to that of ataxic paraplegia, and, as in that disease, with a greater affection of the lateral than of the posterior columns. It differs, however, in the fact that some atrophy of the large nerve-cells of the anterior cornua is common, and also in the constancy with which chronic inflammation of the pia mater is met with, sometimes accompanied by the formation of bony plates in the arachnoid.

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* For some instructive notes on this point in diagnosis see Dreschfeld, 'Brain,' January, 1888.

The *Cause* of the disease appears to be the action, on the elements of the spinal cord, of an organised virus (or of some product of this) which is taken into the system with diseased or unripe maize. Even spirit distilled from such unripe maize may cause the disease. The distribution of the disease in the north of Italy, where the maize is frequently gathered unripe, is significant. The virus has been thought by some (as Lombroso) to be a fungus growing in the maize; by others to be specific micro-organisms of other than fungoid nature, present in the maize, which develop in the system and produce there the toxic agent that has a special action on the nerve-elements (Belmondo). The researches of Tuczek* have clearly shown that the change is a sclerosis of the cord dependent upon some toxic substance occurring in diseased or unripe maize. The effects of the poisons fall, not on the peripheral nervous system, but on the spinal cord, and, as is usual in such conditions (see previous section), the sclerosis induced is symmetrical, and, as will be seen from the illustrations (Fig. 136), in its distribution is very similar to that met with in conditions of anæmia and debility (p. 513).

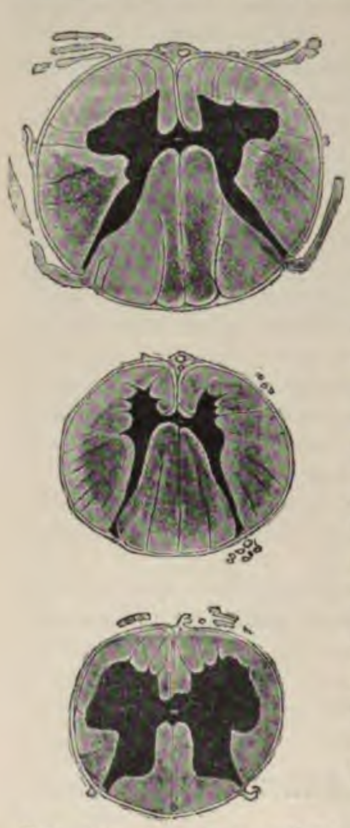

FIG. 136.—Sclerosis in pellagra (Tuczek). The darkly shaded areas indicate the sclerosis.

The *Symptoms* resemble very closely those of ataxic paraplegia. There is weakness in the legs, with increased myotatic irritability going on to spasm; unsteadiness of movement, especially in the early stages, sometimes accompanied by tremor. Sensibility may be normal, or the perception of touch or pain may be increased or lessened. Belmondo, from an important series of investigations,† has arrived at the conclusion that the symptoms depend on a primary systemic degeneration of the nerve-elements of the pyramidal tracts and posterior columns, determined by the influence of the toxic agent. The course of the disease is chronic, but sometimes an intensely acute stage comes on, with symptoms of spinal meningitis,—a condition to which the term "typhus pellagrosus" has been applied. After death in this state there are often found commencing acute myelitis, in addition to the inflammation of the membranes, and also swelling of the intestinal lymphatic glands, with signs of enteritis,—indications of an acute blood-state which justify the view that the affection is, primarily, a toxæmic malady.

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* Tuczek, 'Klinische u. Anatomische Studien über die Pellagra,' Berlin, 1893.

† 'Riv. Speriment.,' 1889–90, xv and xvi.

SCLEROSIS OF THE CORD RESULTING FROM OTHER TOXIC BLOOD-STATES.

During the last few years the effects of toxic blood-states in causing changes in different parts of the nervous system have been widely recognised. The occasional occurrence of sclerotic changes in the spinal cord in association with alcoholic neuritis, not merely as a result of the neuritis, has been already referred to in the chapter on Multiple Neuritis, but the researches of Williamson * have proved the occasional occurrence of sclerosis of the posterior columns in diabetes —a disease with a well-recognised toxic blood-state.

In 1887 Lichtheim † described three cases of pernicious anæmia which, in addition to the usual symptoms of that disease, had symptoms of spinal cord affection. In two the symptoms pointed to a mixed affection of lateral and posterior columns, in the other the posterior columns were thought to be chiefly affected. In the last case no post-mortem examination was obtained, but in the other two distinct spinal cord changes were found, viz. almost complete degeneration of the columns of Goll, and similar but slighter affection of the pyramidal tracts. Lichtheim suggested a toxic blood-state as the probable cause of these changes. Minnich,‡ a pupil of Lichtheim, published a series of similar cases, and supplemented his work by examining the cords of five patients with pernicious anæmia who had had no symptoms of spinal cord affection. He found two sets of changes present, (1) capillary hæmorrhages with apparently miliary sclerosis resulting from them, and (2) a slight sclerotic change in certain parts of the cord as indicated by slighter coloration after hardening in bichromate salts, of the posterior and also of the anterior and lateral columns.

He also found similar changes in the cords of patients the subjects of cachectic and debilitating conditions, such as chronic jaundice, leukæmia, and cerebellar tumour. Within the period that has elapsed since then similar cases have been described by Van Noorden,§

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* 'Brit. Med. Journ.,' 1894, p. 398.

† 'Neur. Cent.,' 1887, p. 236.

‡ Ibid., 1889, p. 662.

§ 'Charité Annalen,' 1891.

[illegible] James Taylor,§ Michell Clark,‖ [illegible] some of the cases having the usual [illegible] others, however, being merely anæmic, [illegible] obvious, but in whom there were dis- [illegible] or mal-nutrition. In 1891 Putnam, of [illegible] group of system scleroses of the spinal cord, [illegible] degeneration occurring in enfeebled [illegible] especially in women." In four cases exa- [illegible] was found in both lateral and posterior [illegible] he also found disintegration of cells in the [illegible] are no doubt similar in origin to those [illegible]

[illegible] majority of cases the onset of the spinal cord [illegible] preceded by a period of bad health, more or less [illegible] condition leading to impairment of vitality. Thus [illegible] of blood immediately preceded the anæm[illegible] affection was afterwards added. Difficu[illegible] is first complained of, and one leg may [illegible] more severely than the other. Sharp lancing [illegible] lightning pains of tabes, are occasionally pr[illegible] necessarily painful, are frequent. The [illegible] normal or diminished, and there m[illegible] In some cases, however, they a[illegible] may be present on one side and not on th[illegible] as sometimes happens, and som[illegible] picture presented is that of ataxic parapl[illegible] increases until the patient is unable to [illegible] impairment may occur, considerable ri[illegible] jerks, in cases in which they were [illegible] The arms also may be involved [illegible] movements at the wrist and el[illegible] of canine chorea, and probably [illegible] come to discuss the morbid a[illegible] superadded, and bedsores, cystiti[illegible] later lead to a fatal issue.

[illegible] enumerated there may of co[illegible] anæmia—such as reductio[illegible] enlargement of spleen, a[illegible] But, as has been stat[illegible] condition under notice.

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[*] [illegible] Med. Wochenschr.,' 1892.

[†] 'Arch. f. Psych.,' xxv, 1893.

[‡] 'Brain,' 1894.

[§] 'Med.-Chir. Trans.,' 1895.

[‖] 'Brit. Med. Journ.,' 1897.

[¶] 'Lancet,' 1898.

[**] 'Journ. of Nerv. and Ment. Dis[illegible]

MORBID ANATOMY.—The changes found in the spinal cord are curiously uniform (Fig. 137). In the cervical region they are usually

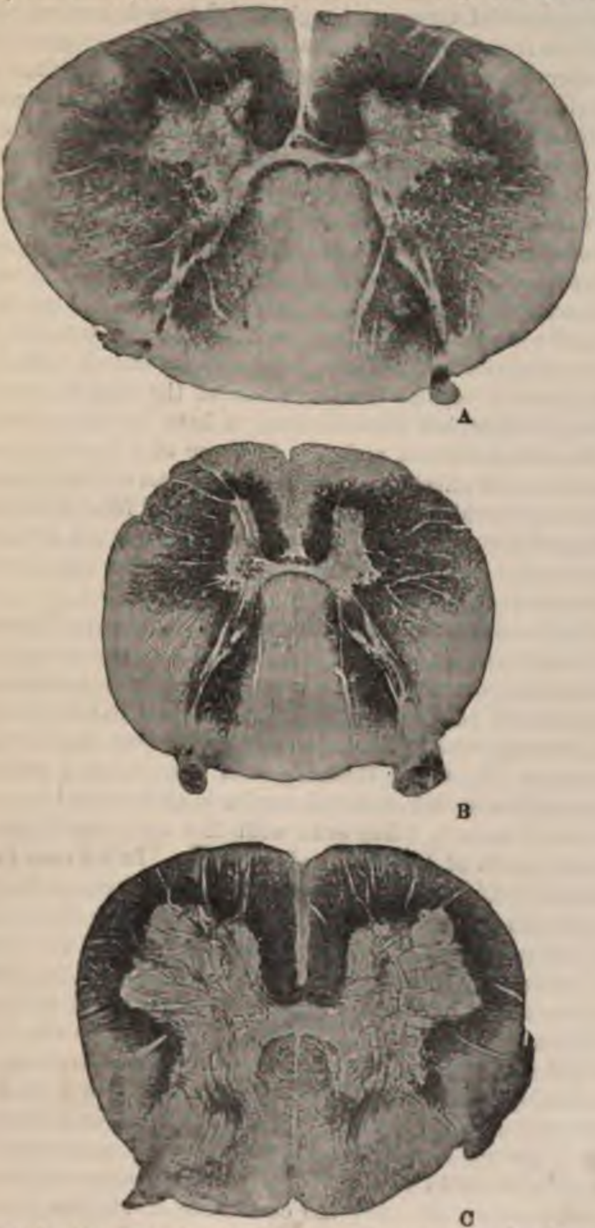

FIG. 137.—Showing sclerosis occurring in a case of profound anæmia. The sclerotic areas are decolourised. Note in the lumbar cord the comparative escape of the septo-marginal and cornu-commissural tracts. A, cervical. B, dorsal. C, lumbar.

Eisenlohr,* Nonne,† Bowman,‡ James Taylor,§ M
Risien Russell,¶ and others, some of the cases ha
symptoms of pernicious anæmia, others, however, bein
some in whom anæmia was not obvious, but in whom
tinct evidences of debility or mal-nutrition. In
Boston,** described "a group of system sclerosis
associated with diffuse collateral degeneration o
persons past middle life, especially in women."
mined post mortem sclerosis was found in both
columns of the cord, and he also found disinte
grey matter. These cases are no doubt simi
described by Lichtheim.

SYMPTOMS.—In the majority of cases the o
symptoms is usually preceded by a iod of l
prolonged, or by some condition le g to imp
in one case, a profuse loss of blo mmediate
to which the spinal cord affect was after
or uncertainty in walking is fir xplained
affected earlier and more sever han the
pains, suggesting the lightnin s of tab
and paræsthesiæ, not neces painful,
jerks are usually at first n or dimi
difference on the two sides. some
lost, or the reflex may be t on o
When they are exaggerated ometim
present, the clinical picture nted i
weakness gradually increases til the
bed, then sensory impairment may
come on, and the knee-jerks, in cases
be no longer obtainable. The arms
case curious rhythmical movements
sent, suggesting those of ca ine ch
as we shall see when we co e to d
tractions may be supera ed, a
exhaustion sooner or later ad to
Besides the symptoms en umera
symptoms of profound anæmia
and number of corpuscles, enlarg
the retinæ and elsewhere. Bu
is not constant in the condition

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ll exten-
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rior septum
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the so-called
tracts in the
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Clark's column, n
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ges are present, a
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is apparently a sin
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marked increase in
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In no case i
any hæmorrhage be
however, that I
anæmia, in whic
during life,
different parts of
of this will be al
under considerat
to doubt that
result of a toxic
of at least mal-
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* 'Deut. Med.
† 'Arch.
‡ 'Brain,
§ 'Med.
‖ 'Brit.
¶ 'Lanc
** 'Jour

indication that the blood is probably the vehicle by which the *materies morbi* is brought to its destination; and it is very significant that the changes in the spinal cord in pellagra, an admittedly toxic disease, are practically identical in distribution with those in the conditions under notice. The nature of the poison we do not know, and it cannot be said with certainty whether it acts directly on the nervous elements, or by first producing disease of the blood-vessels. In many cases the blood-vessel walls are found thickened, in some instances to the point of occlusion, and the occurrence of hæmorrhages in the retinæ, and elsewhere under similar conditions, suggests that diseased vessels and consequently altered blood-supply may give rise to interference with the nutrition of the cord, and so produce a sclerotic condition. Such an explanation is not probable. Yet there seems no reason to doubt that in some of the cases the occurrence of small hæmorrhages in the cord, similar to those that occur in the retinæ in pernicious anæmia, may be an element in producing the changes which we meet with. The observations of Minnich and others already alluded to, on the cords of patients suffering from pernicious anæmia, demonstrate the occurrence of such hæmorrhages, and of small foci of degeneration as the result of them, in those cases in which, it is true, no special symptoms of cord affection were noted during life. But if such hæmorrhages occur in these cases, it seems likely that they are not altogether absent in cases in which disease of the cord is more pronounced. And it must be confessed that in some cases the difference between two sections of the cord, within a short distance of each other, is greater than is to be satisfactorily accounted for, except on the supposition of some local condition to give rise to the difference. Such hæmorrhages would of course be no part of the ultimate pathology of the affection, which, as we have said, is almost certainly toxic; but they might account for some of the changes visible in the spinal cord, especially those which constitute a departure from true symmetry.

The view of Nonne and others, that the condition is really one of disseminated myelitis, offers many difficulties. The absence of any small-celled infiltration such as is found in myelitis, the absence also of such ascending and descending degenerations as foci of inflammation would produce, seem to constitute insuperable difficulties to the acceptance of such a view. That the condition is to be accounted for by patches of change, hæmorrhage perhaps, in the grey matter, is most unlikely. In many cases no change is found in the grey matter; when any change is present it is slight and non-symmetrical, and one striking feature is the escape from degeneration of the endogenous fibres of the cornu-commissural and septo-marginal tracts of the cord (see p. 218), tracts which connect the grey matter at different levels, and which would presumably suffer conspicuously in any primary affection of that tissue. So that on all grounds the change is to be regarded as probably of the nature of a parenchymatous degeneration of the nerve substance, with secondary overgrowth and hardening of the connective

wide-spread, and are found in anterio
the part of the anterior column chiefl
being the pyramidal tracts, although
these in either region. In the dorsal
sive, but there is a gradual diminut
lumbar region the sclerosis is much le
in character. In this region the part
commissure is usually spared, as also
—the septo-marginal tract of prob
alluded to (p. 218). It is well seen i
It is interesting to note that the other
is spared probably also consists of
cornu-commissural tract. The sclero
lumbar cord usually becomes restri
close to the posterior root. These de
to extend upwards beyond the lower
and although slight but distinct
cells of the anterior horn, and also
definite evidence of affection of the
in the few cases in which changes
more than probable that in some
it may be expected that they
regularly and carefully examined.

The change which constitutes
thickening and increase of the
which have disappeared. The
number of vessels in the
well seen in most cases in
lumbar regions. In one of
work of small thin-walled
with thickened walls." But
marked infiltration of the
these degenerations have
in any of the affected tracts
and Minnich describe in
ptoms of spinal cord
rotic foci the result of
cord, The importance
discussing the patholog

PATHOLOGY.—There
condition presen in
the blood. Its origin
debility, its pr
from pernicio
similar distri
probability.

definitely anæmic cases iron and arsenic are clearly indicated, and they are so probably in all cases. Bone marrow does not seem as yet to have received a trial, but that also in combination with the drugs mentioned would possibly be found to have a good effect. Cystitis, bedsores, and contractions must be treated if they arise. The pains may be severe and necessitate morphia, but the need for this may be lessened by injections of cocaine, as recommended in the chapter on Tabes.

"HEREDITARY ATAXY"

(FRIEDREICH'S DISEASE, HEREDITARY ATAXIC PARAPLEGIA).

The so-called hereditary ataxy is a form of ataxy, or rather of ataxic paraplegia, depending on combined posterior and lateral sclerosis, which occurs in families, and differs further from the common forms of tabes and of ataxic paraplegia in the early age at which it always commences, and in the presence of certain additional symptoms. It is often termed "Friedreich's disease," because this physician first described the characteristic features of the malady and the lesion in the posterior columns.[*]

The dependence of the disease on a congenital tendency is clearly shown by its usual occurrence in families. But direct inheritance has been traced in only a few instances. In one, the mother of the family affected, and *her* mother, both suffered from the disease. That a general neuropathic inheritance is also sometimes effective is shown by cases in which there is a history of other neurotic diseases, such as insanity or epilepsy, in collaterals or ancestors. Two brothers who were affected had another brother epileptic, and a sister suffered from repeated attacks of chorea. Alcoholism in parents has been supposed to be influential, but the evidence of this is not strong. Consanguinity of parents has existed in some instances, and has doubtless intensified the morbid tendency in these cases.

The family tendency of the disease is shown by the affection, in most instances, of brothers and sisters. Thus sixty-five cases were distributed in nineteen families, giving an average of rather more than three to each. The number affected in one generation has varied from two to eight. In the case in which the mother and grandmother

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[*] Friedreich's first account was given to a medical society in 1861, and published in 'Virchow's Archiv' (Bde. xxvi and xxvii) in 1863. Further cases were published by him in 1876 (ib., Bde. lxviii and lxx), and a collection of 57 cases is given by Dr. Everett Smith, 'Boston Med. and Surg. Journal,' Oct. 15th, 1885. See also Ormerod, 'Brain,' Jan., 1888; Dejerine, 'Méd. moderne,' 1890, No. 25, and 'La Sem. Méd., 1890, Nos. 11, 12; and especially Soca, 'Thèse de Paris,' 1888 (who has collected 165 cases—many, however, doubtful), and Ladame, 'Brain,' 1890, pt. iii, where a full bibliography will be found; see also Mackie Whyte, 'Brain,' 1898.

[illegible] children were also affected, making te [illegible] also been recorded, and they seem [illegible] the nature of the affection has not b [illegible] isolation. In many of these cases [illegible] this feature extends beyond [illegible] child, or the only one of the sex in [illegible] the only daughter, and her fo [illegible] other hand, it is often to be not [illegible] cases of Friedreich's disease occur a [illegible] family of fifteen, in which three membe [illegible] however, transmitting the conditio [illegible]

[illegible] equal liability; males slightly pre [illegible] thirty females). In some families th [illegible] but in others the disease has shown [illegible] sex. Thus in one family of ninetee [illegible] the seventeen females escaped. In [illegible] by Musso, a brother and sister (the [illegible] mother) married healthy individuals; [illegible] affected out of seven living children; [illegible] three sons were diseased. A curious fact [illegible] and four of the sister's children were [illegible]

[illegible] first symptoms are recognised has varied [illegible] years.* The seventh and eighth years [illegible] disease most often begins; and next comes [illegible] twelve to sixteen. In isolated instances [illegible] than in the grouped cases. It begins [illegible] than in females, and often commences about [illegible] family.

[illegible] be traced; preceding acute diseases [illegible] but can scarcely have done more.

[illegible] and chief manifestation of the disease is a [illegible] coordination, first in the legs and afterwards [illegible] scarcely ever occur, but cramp is occa- [illegible] The ataxy is shown by unsteadiness in [illegible] at first slight, but slowly increasing, until the [illegible] wide apart in standing, and the patient reels in [illegible] the influence of alcohol. The feet are not [illegible] unless when an unusually long step is taken. [illegible] a considerable increase in the unsteadiness [illegible] it has but little influence. Children often first

* [illegible] by Everett Smith, the father presented symptoms of [illegible] at the age of sixty-six, coming on gradually after an attack of [illegible] exposure to cold. In the age of the sufferer this case [illegible] of the disease, and is not included in the figures given. [illegible] coincidence, or may show that a latent predisposition may [illegible]

show the affection by the readiness with which they stumble and fall. The impairment of movement in the arms is of a similar character, but usually commences some time after that of the legs. There is irregularity in the voluntary movement of the arms and fingers, and the ataxy has often a distinctly jerky character. In most cases the power of the muscles is at first unimpaired; their nutrition is good, but the myotatic irritability is lost. In most cases the knee-jerk has been found absent as soon as the patient came under observation; in one case it disappeared after the other symptoms had set in; in another it was normal on one side, absent on the other. In several there has been no change long after all the other characteristic symptoms had manifested themselves; very rarely it has been increased, chiefly in untypical cases.

As the disease progresses some jerky irregularity develops in the movements of the neck and head, so that the head presents slight unsteady movements, sometimes like an irregular tremor, sometimes closely simulating chorea. Articulation is also impaired; syllables are elided; there is a blurred, somewhat explosive utterance, and there may be with this an occasional hesitation. There is no twitching of lips, but occasional jerky movements have been noted in the tongue. The affection of speech is not often an early symptom. It may not be noticed until three, five, or ten years after the onset of the other symptoms. In most cases (but not in all) there is nystagmus when the eyes are moved laterally or upwards. Sometimes the movement is slower than in most forms of nystagmus, and it is rarely present when the eyes are at rest, directed straight forwards. This symptom may come on after those in the limbs, but careful examination will often reveal it in the early stage. Paralysis of the ocular muscles is very rare; in one case there was strabismus with double vision,* and slight diplopia sometimes exists for a time and passes away. Optic nerve atrophy never occurs. The pupils are usually normal, but several cases have been recorded in which there has been loss of the light reflex. Whenever this condition exists evidence of hereditary syphilis should be most carefully sought for, as it is probable that many of the cases in which this loss was met with were cases of true tabes, with some involvement of the lateral columns occurring in young persons, and should not be classed with the disease in question.

Sensory symptoms are very variable. Lightning pains and any severe pains are extremely rare, though in one or two recorded instances they have been severe; but slight dull or rheumatoid pains in the legs are not uncommon. Sensibility has been quite normal in many cases even of severe degree; in others there has been slight anæsthesia in the legs, early in some instances, late in others. In one case there was delay of conduction, such as is met with in tabes;† rarely sensibility to pain and temperature is impaired. The sense of posture of the limbs has been found normal in several cases in which

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* Charcot, 'Prog. Méd.,' 1887, No. 23.

suffered, an uncle and seven children were also affected,
one family. Isolated cases have also been recorded, and
to be uncommon. Probably the nature of the affection
suspected on account of the isolation. In many of
judge from personal observations), this feature ext
disease; the patient is an only child, or the only one
family. Thus one patient was the only daught
brothers were healthy. On the other hand, it is
that the families in which cases of Friedreich's
unusually large, as in one family of fifteen, in w
were affected; another unaffected, however, transm
to two out of four children.

The two sexes present nearly equal liability
ponderate (thirty-five males to thirty females).
two sexes have suffered equally, but in others
marked tendency to affect one sex. Thus in
the two males suffered, and the sevente
another instance, recorded by Musso, a
offspring of a melancholic mother) mar
the brother had three daughters affected
and of the sister's children three sons we
is that three of the brother's and four
born dead.

The age at which the first sympt
between two and twenty-four years.*
of life are those in which disease mo
the period of puberty, from twelve
the onset is generally later than
somewhat earlier in males than in f
the same period in the same family
Immediate causes can rarely b
may have facilitated the onset, bu

SYMPTOMS.—The first and chief
gradual impairment of co-ordin
in the arms. Initial pain
sionally complained of. The
standing and walking, a first
feet have to be placed w
walking like one unde
often raised too high,
Closure of the eyes ca
in most cases; in sor

marked lateral curva
stary.

weakness in the flex
The los
its to ab
n the la

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* In the family reco
ataxic paraplegia at t
rheumatism produced
stands alone in the b
The case may be a
persist through life.

disease, attended with only trifling depression of the electrical contractility; in two recorded cases, however, a brother and sister, there was great atrophy of muscles with altered electrical reactions.* Lateral curvature of the spine, and talipes equinus or equino-varus of the feet (see Figs. 138 and 139), are common as later results of the muscular weakness, developing under the influence of posture; and contraction of the flexors of the knees has also resulted.

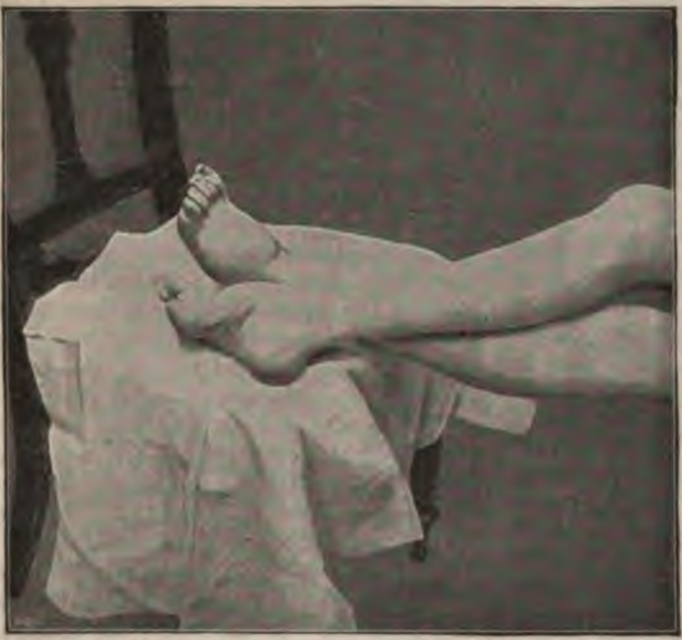

FIG. 139.—Showing the characteristic deformity of the feet in Friedreich's ataxy.

Visceral crises do not occur. Frequency of pulse has been noted, however, in many cases, and curious vaso-motor symptoms (flushing, œdema, sweating, polyuria, and salivation) were present in one of Friedreich's cases. A basic cardiac murmur is of very frequent occurrence, possibly associated with the anæmia which is frequently present. There is no mental change that can be regarded as part of the disease, although imbecility has co-existed (Power).†

The isolated cases may correspond closely to the type, presenting the same nystagmus, weakness, and unsteadiness; many of these cases, however, differ somewhat, although sufficiently characteristic in their main features to justify their inclusion Speech more often escapes. I have once met with a congenital lisp. In some the symptoms are slight, and have developed late. A difficulty in micturition may precede other symptoms. There are also cases, sporadic and grouped, in which coarse tremor is conspicuous, and the condition might be regarded as one of simple tremor, were it not for the presence or development of other symptoms. For instance, one man had clipping speech "all his life;" between forty and fifty difficulty in writing came on, owing to a peculiar spasmodic tremor in both arms, brought on also in the legs by exertion; at fifty unsteadiness on walking was added. There was a history of a similar affection in several relatives.

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* Dejerine, 'Méd. mod.,' 1890, No. 25.

† An excellent analysis of the symptoms, &c., is given by Crozier Griffith ('Trans. Coll. Phys. Philadelphia,' 1888; and by Ladame ('Brain,' 1890, pt. 52), and Mackie Whyte ('Brain,' 1898).

The rate of progress of the malady varies much, even in different members of the same family. One, in whom the disease commenced last, may be unable to walk, while another who suffered sooner may be still in the early stage of the disease. Now and then the disease is stationary for many years. The duration of the malady is correspondingly variable. It is always long, even more than thirty years, and in many instances it has not apparently shortened life. On the other hand, death may occur at the end of ten or twelve years. The end has generally come from intercurrent affections, rarely from exhaustion. Acute myelitis has also caused death (Everett Smith).

A few years ago, from a consideration of the results of his own observations and those of others, Marie* concluded that there exists a type of disease in many points resembling Friedreich's ataxy, but differing in several important particulars This he called *hereditary cerebellar ataxy*, and he regarded the symptoms as the result of some congenital defect in the cerebellum. In the ataxy, the articulatory difficulty, the frequent presence of nystagmus, and the fact that the disease might occur in more than one member of the same family, the condition closely resembled Friedreich's disease, but striking differences were manifested in the occasional presence of the Argyll Robertson pupil, the not unusual occurrence of optic atrophy, and the invariable exaggeration of the knee-jerk. The onset of symptoms also was usually much later in this disease. The remarkable series of cases described by Dr. Sanger Brown† probably belong to this class. They are characterised by the occurrence of a similar condition in many members of the same family throughout several generations, viz gradually increasing weakness and inco-ordination in the legs, with marked tremors in the head and body, impaired articulation, exaggerated knee-jerk, occasional ankle-clonus, and frequently optic atrophy. There was no nystagmus and no deformity. Nonne and Menzel have also described similar conditions, but in Nonne's cases the ocular movements were impaired, and there was mental weakness. Gee,‡

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* 'La Semaine Médicale,' 1893.

† 'Brain,' 1892, Summer.

‡ 'St Bart.'s Hosp. Rep.,' vol. xxv.

Tooth,* and others have also described a family disease characterised by symptoms of spastic paraplegia (lateral sclerosis).

Pathological Anatomy.—The lesion in hereditary ataxy is that of ataxic paraplegia and of tabes combined. There is degeneration in the lateral column, often also in the anterior column, such as occurs in ataxic paraplegia but the sclerosis of the posterior column is more intense than in that disease, and it is also more extensive, especially in the lumbar region It is similar to the degeneration of tabes, and agrees with this also in that the posterior nerve-roots are usually affected, whereas in ataxic paraplegia they almost invariably escape.

The distribution of the lesions shows a close correspondence in different cases, as will be seen by a comparison of the figures here given, which are from three different cases of the disease. The sclerosis

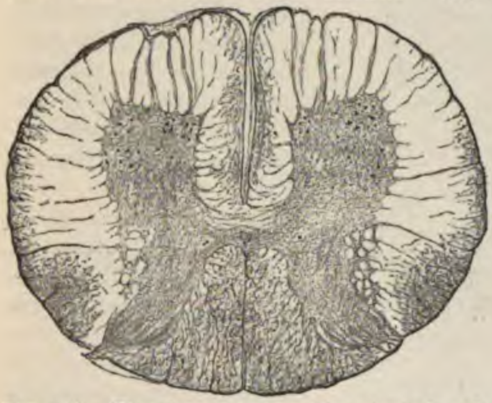

Fig. 140.—Hereditary ataxy; section of spinal cord at first lumbar segment. Sclerosis of the whole of the posterior columns except a narrow zone adjacent to the neck of the horn. Degeneration also of the lateral pyramidal tracts, and, in front of this, slight degeneration in the superficial layer of the lateral column. Adjacent to the anterior median fissure there is also a zone of sclerosis of the anterior pyramidal tract, which extends, in this cord, into the lumbar region.†

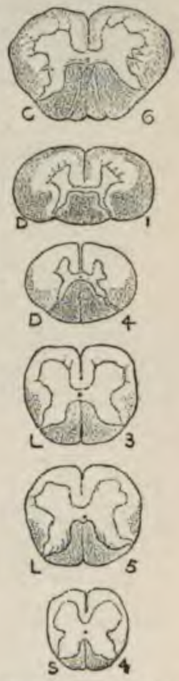

Fig. 141.—Hereditary ataxy; distribution of degeneration in the white columns, indicated by the dotted shading. (After Friedreich.)

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* 'St. Bart.'s Hosp. Rep.,' vol. xxvii.

† I am indebted for this section to Dr. Everett Smith, who has published the case in the 'Boston Med. and Surg. Journal,' Oct. 15th, 1885. The patient was one of three sisters affected with the disease, the brothers being healthy. The father suffered from ataxic paraplegia late in life. In the case from which the drawing is made the inco-ordination began in the legs at the age of nineteen, and in the arms soon afterwards, and was quickly followed by loss of power, which gradually increased to almost universal paralysis, with muscular contractures (talipes equino-varus, &c.) and considerable loss of sensation. There were nystagmus, affection of articulation, and some mental dulness. Death occurred at the age of forty.

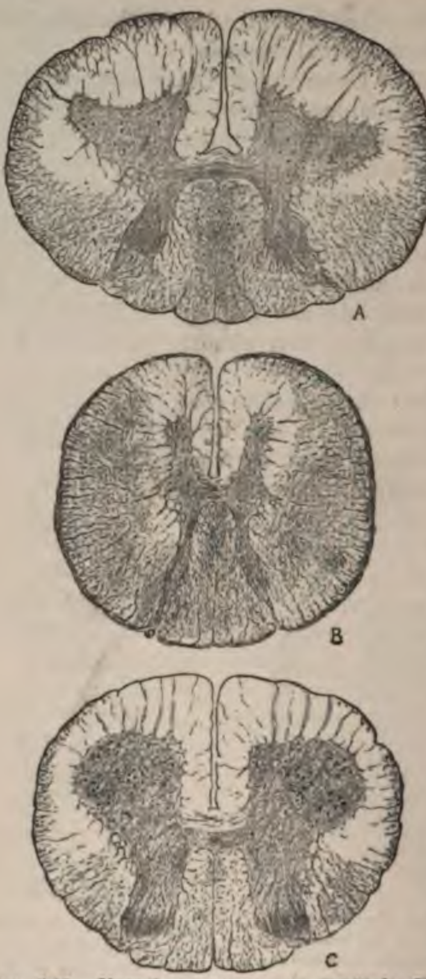

FIG. 142.—Hereditary ataxy. A, cervical; B, dorsal; C, lumbar regions of the cord. The posterior columns are sclerosed in their whole extent, except in the vicinity of the neck of the horn in the cervical and lumbar regions, the degeneration being rather less intense in the lumbar than in the other parts. In the antero-lateral columns there is an annular zone of sclerosis in the periphery of the cord, widening in the region of the pyramidal tract. In front of this tract the zone is widest in the dorsal region, and trifling in degree in the lumbar. The grey matter is but little affected.*

of the posterior columns may be complete throughout the cord, with the exception of a narrow band near the neck of the cornu, which always remains but little damaged. In the lumbar region it is usually intense up to the head of the horn and the posterior surface of the cord; occasionally, however, it is somewhat less intense in this region than it is higher up, extending, nevertheless, into the root-zone. In the cervical region the sclerosis may also be general, or it may be greatest in the root-zone and in the posterior median columns. The cervical root-zone never escapes, as it often does in tabes.

The degeneration of the lateral columns always involves the pyramidal tracts, and is most intense in their position. It is not, however, limited to them. It usually extends outwards to the periphery of the cord, even where the pyramidal tract does not reach the surface, thus involving the direct cerebellar tract; and it also extends forwards at the surface, so as to [illegible] stitute a band of [illegible] sclerosis, [illegible] thickness [illegible] of th[illegible] cas[illegible] the inner part of the anterior column there may be a distinct area of degeneration in the position of the anterior pyramidal tract. This is seen in Fig. 140, in which this tract extends unusually low in the cord, and is distinctly degenerated in the upper part of the lumbar enlargement. Atrophy of the posterior vesicular column has been found associated with that of the direct cerebellar tract. Slight abnormal appearances have been seen in the nerve-cells of the anterior cornua in some cases, but as a rule these are normal, and the grey matter presents no other disease. It is very likely, however, that future observations will reveal the occurrence of changes in the nerve-cells of the posterior horns, such as are met with in tabes.

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* For the sections from which the drawi[illegible] (who prepared them) and the late Dr. Mox[illegible] Guy's Hospital. The case (full details of [illegible] the 'Guy's Hospital Reports') is one of [illegible]

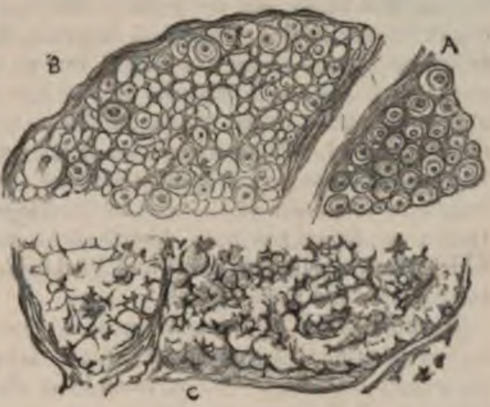

FIG. 143.—Hereditary ataxy; degeneration of posterior nerve-roots (from the same case as Fig. 142). A, normal anterior root, for comparison. B, posterior root, partially degenerated; a few normal nerve-fibres are seen, but most of the spaces which should contain nerve-fibres are empty. C, posterior root, totally degenerated, probably by a more acute process than that of B, since there is some destruction of the septa and increase of amorphous connective tissue. The products of degeneration are rendered invisible by the mode of preparation. (Compare C with the similar changes in a totally degenerated peripheral nerve shown in Fig. 59 B, p. 165.)

The pia mater over the posterior columns has been found thickened. The posterior nerve-roots usually present some degeneration of their fibres; this may be partial (Fig. 143, B), or may amount to total destruction of the fibres (as at C). The peripheral nerves in the limbs have been found normal.*

Some general shrinking and induration of the pons and medulla were found by Friedreich, with atrophy of the cells of the post-pyramidal nucleus and some degeneration of the restiform bodies, but none of the anterior pyramids. Corpora amylacea were present in the hypoglossal nerves.

Atrophy of the cerebellum, pons, and olivary bodies was found in one case; the cells of Purkinje were diminished in numbers, but showed no sign of atrophy or degeneration.† There was also degeneration of the hypoglossal and facial nuclei.

This defective cerebellar development is the condition supposed to

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sister) which I reported some years ago to the Clinical Society ('Clin. Soc. Trans.,' vol. xiv, 1881, p. 1).

* Friedreich, 'Virchow's Archiv,' Bd. lxx, p. 145.

† P. Menzel, 'Arch. f. Psych.,' Bd. xxii, H. 1, p. 160.

underlie the manifestations of the condition already alluded to as the hereditary cerebellar ataxy of Marie. In another case * the anatomical condition revealed the absence of cord degeneration, but the cerebellum was small, and the cells of Purkinje deficient. In the only one of Sanger Brown's examined, however, there were sclerotic changes in the lateral and posterior columns, but no marked cerebellar changes were present, although the cells of Purkinje are said not to have been over-abundant. A case, similar in clinical conditions, was many years ago described by Fraser and Coats of Glasgow, and in this also there was no cord change, but marked atrophy of the cerebellum, and a defective development of the cells of Purkinje. It is of some interest that a condition has been described in cats congenitally ataxic, in which the cerebellum is small, and Purkinje's cells absent, or defective in character and number.

Pathology.—The disease apparently occupies a clinical and pathological position between the combined sclerosis described as ataxic paraplegia and simple tabes, resembling the former in the common affection of the lateral and posterior columns, and the consequent weakness with ataxy—resembling it also in the imperfect limitation of the changes, and in the intensity of those in the middle of the posterior columns in the dorsal region; but it differs from this, and resembles tabes, in the degree of affection of the posterior columns in the lumbar regions, the extension in slight degree into the root-zones, and the affection of the posterior roots: with these the loss of the knee-jerk and the affection of sensation are apparently connected. The isolated cases, occasionally met with, present the same combination of spinal symptoms. The precise origin of the affection of articulation and of the nystagmus has not yet been traced.

The age at which the disease commences, long before the period at which ordinary degenerations occur, and its family grouping, suggest that its ultimate cause is a congenital tendency of development, by which the affected elements have a briefer period of vital endurance than the other tissues of the organism. Möbius has suggested that there is an actual arrest of development of these structures, but it is evident that their development suffices for perfect function during the early part of life, and that their functional capacity undergoes subsequent failure, which can only be due to a process of structural change. It is possible that we have here, again, a double morbid process, and that a tendency to early degeneration in the nerve-elements is associated with a converse tendency to overgrowth of the interstitial tissue, analogous to that which we have in the muscles in pseudo-hypertrophic paralysis (*q. v.*). In this connection it is important to note that in one case the neuroglial overgrowth suggested a pure increase of the normal tissue, rather than a result of inflammation; and the peripheral sensory nerves, although not degenerated, contained a considerable number of embryonal nerve-fibres in the fasciculi.* Dejerine would regard the neuroglial increase as the sole element in the morbid process; but the fact last mentioned, as well as the preponderant affection of certain tracts, makes it improbable that this exclusive view is correct.

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* Nonne, 'Arch. f. Psych.,' xxii, 1891.

Although we have here only to consider hereditary cerebellar ataxy and similar conditions, in relation to Friedreich's ataxy, it may be stated that in this disease also we probably have to deal with a developmental defect or perversion, as has been indicated in considering the morbid anatomy. And the same may be said of the family form of spastic paralysis. It might already seem as if we had a series to deal with, with Friedreich's ataxy at one end and family spastic paralysis at the other, the intermediate space being occupied by cases like those of Sanger Brown, in which spasticity and ataxy are combined, and in which the underlying anatomical condition as regards the cord is one of sclerosis affecting lateral and posterior tracts fairly equally, while in Friedreich's disease we have to deal with a condition in which the posterior columns suffer most, and in family spastic paralysis with one in which the lateral columns may be exclusively affected.

DIAGNOSIS.—In most cases the inco-ordination is sufficiently predominant to suggest that the disease is a form of ataxy, and the unsteadiness of the head, the affection of articulation, the nystagmus, and the age at onset, suffice for the distinction from ordinary tabes. The common form of ataxic paraplegia bears a close resemblance to this disease, a resemblance that is more than superficial, although the excessive knee-jerk and foot-clonus, almost always present in ataxic paraplegia, are usually absent in hereditary ataxy; much spasm is always absent, and nystagmus is common. The difference in the state of myotatic irritability is, as we have seen, not absolute; cases of combined lateral and posterior sclerosis occur, of tabetic type, in which the knee-jerk is lost, and in at least one case of hereditary ataxy the knee-jerk has been excessive. The age of onset and family multiplicity may decide the question, but the isolated cases, which are by no means rare, commencing soon after puberty, are not distinguishable from the hereditary disease; they seem to be analogous to the isolated cases of pseudo-hypertrophic paralysis. A greater difficulty is presented by cases of slight tabes in children, the subjects of inherited syphilis, in whom it is not uncommon for some weakness of the legs to co-exist, or speech to be disordered, in consequence of some early lesion of the brain. The loss of the iris-reflex should suggest inherited syphilis, and other indications of this will often decide the nature of the case.

Disseminated sclerosis presents inco-ordination, nystagmus, and impaired articulation, but the ataxy of the arms differs in the wide range and violent character of the disordering jerks which characterise this disease; while simple unsteadiness of the legs is very rare, and the course of the malady is more rapid. The affection of speech differs from that of hereditary ataxy in being simply "staccato," with undue separation of syllables, in the marked cases. In many instances, however, the articulatory defect is similar in the two diseases. The cases are always isolated.

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* Anscher, 'La Semaine Méd.,' 1890, No. 32.

Cerebellar tumour and hereditary ataxy can hardly be confounded, in spite of the fact that the unsteadiness in walking is very similar in the two diseases; the common affection of the arms in the one, and the conspicuous head symptoms (severe pain, optic neuritis, &c.) of the other, sufficiently distinguish them. It is very common for the tremor of the head to be at first ascribed to mere "nervousness," and also for the isolated form, in girls, to be regarded as hysterical. The presence of nystagmus should at once decide the question; it is absolute proof of definite disease.

PROGNOSIS.—The prognosis in every case is very serious, since the disease, being a developmental affection, is essentially progressive; but life may be prolonged for many years, and in slight cases the malady may interfere comparatively little with the patient's occupation. Thus I have known a man, with very marked symptoms, follow his business as a tradesman for many years. The only guide to individual prognosis is the observed rate of progress, which has little relation to the age at which the symptoms commence.

TREATMENT.—As in other diseases that depend on a congenital tendency, treatment is almost powerless. The measures recommended for ordinary locomotor ataxy (apart from those suggested by the special symptoms of tabes) are those most suitable to the hereditary form. Arsenic, phosphorus, and nitrate of silver deserve a trial; now and then they seem to check the progress of the disease for a longer or shorter time, but its individual tendency determines its course, almost irrespective of treatment.

SIMPLE SENILE PARAPLEGIA.

This name seems the most unobjectionable for a condition, somewhat rare, which appears hitherto to have been unrecognised.* It is confined to late life, occurring in those over 40, and especially over 50. Its characteristic is simple weakness of the legs, with some slowness of movement, but without wasting, sensory disturbance, or reflex alteration. The knee-jerk is normal, and there is no foot-clonus. The malady develops very gradually and is slowly progressive, although it seems seldom to attain such a degree as to prevent standing. The condition of the legs resembles that in cases of paralysis agitans without tremor, in which the malady is manifested only by weakness, and stiff slowness of movement of the limbs, face, and trunk. Cases are met with in which the condition of the legs above described is associated with slight symptoms in the arms and face, such as characterise these cases of paralysis agitans. Thus senile paraplegia is probably a partial development of the morbid process of that disease; instead of being general, it is limited to the structures for the legs. If, as the condition seems to suggest, paralysis agitans depends on peculiar changes in the nutrition and function of the motor cells of the cortex cerebri (which impair the power and alter the action of the cells, but do not usually go on to such destructive alteration as entails secondary degeneration of the pyramidal fibres), we can readily conceive that such a process may be sometimes limited to the leg-centres, and may cause the symptoms. If so, the malady is a cerebral and not a spinal one, but it is mentioned here on account of its paraplegic features, and their resemblance to those of disease of the spinal cord. Degenerative changes may, however, be various in kind and degree, especially in late life; cases occur, similar to these, but with myotatic excess connecting them with the destructive changes of lateral sclerosis, while others present such symptoms as nystagmus or impeded articulation—evidence that the altered nutrition is wider in its range.

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* A note upon it by myself appeared in the 'Centralbl. f. Nervenkr.,' 1890.

CHRONIC SPINAL MUSCULAR ATROPHY.

(PROGRESSIVE MUSCULAR ATROPHY; WASTING PALSY; AMYOTROPHIC LATERAL SCLEROSIS; CHRONIC POLIÒ-MYELITIS).

The disease which has long been known by the names "progressive muscular atrophy" and "wasting palsy" is characterised by slow wasting of the muscles, beginning in some one part, and usually spreading and increasing until it is wide in extent and extreme in degree. The changes in the muscles depend on changes in the spinal cord, a slow degeneration of the ganglion-cells of the anterior cornua, accompanied by a similar degeneration in the motor nerve-fibres which spring from the cells. With this degeneration of the cells and peripheral fibres there is usually also associated a degeneration of the pyramidal tracts in the cord, sometimes, at least, to be traced up to the motor cortex of the brain. According to current views, there is a decay of the spinal motor neuron, and often also of the upper neuron, which has its nutritional centre in the cells of the motor cortex.

Chronic muscular atrophy was separated from primary paralysis by Sir Charles Bell, and was afterwards described more fully by Aran *

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* Aran, "Recherches, &c., sur Paralysie Musculaire Atrophique," 'Arch. gén. de Méd.,' 1850.

the course of [illegible] Cruveilhier regarded it as a spinal disease.
differs from this [illegible] progressive muscular atrophy") thought
undue separation [illegible] of the muscles, a view that was after-
stances, however [illegible] and subsequently also by Friedreich. This
eases. The [illegible] by a theory that the wasting was due to a
Cerebellar tu[illegible] Luys‡ first discovered the disease of
in spite of the [illegible] spinal cord, and Charcot especially associated
the two disease [illegible] of the ganglion-cells. When the con-
the conspicuous [illegible] demonstrated, it was thought that every
the other, suffic[illegible] atrophy was of spinal origin; but this view
tremor of the [illegible] has been shown that there are forms of
also for the iso[illegible] purely local and idiopathic. The morbid
presence of [illegible] spinal cord. Atrophy of the nerve-cells
absolute proof [illegible] nerves arise often accompanies that of
PROGNOSIS.—[illegible] bulbar paralysis" is then associated
disease, being [illegible] atrophy:" it often comes on after the dis-
life may be p[illegible] and ends life; occasionally it initiates
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is the obser[illegible] manifested only by the wasting of the mus-
which the [illegible] type;" in the other there are, in parts
TREATMENT[illegible] atrophied, the indications of degeneration of
tendency, [illegible] the cord. In the latter cases, Charcot re-
for ordinary [illegible] of the pyramidal tracts as the primary
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form. A[illegible] first class "protopathic," and those of the
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or shorte[illegible]" In Germany, however, and especially by
almost [illegible] expressed as to the validity of the dis-
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conti[illegible] M.A., M.D., Glasgow, 1895, an admirable account of the
out [illegible] of all these diseases. To this book the reader may be
[illegible] regarding the subject than can be given here, not only
[illegible] but to all others of a degenerative character.

every gradation to subacute polio-myelitis in one direction, and perhaps also to polyneuritis in another. Attempts are being made to classify these, but types may be multiplied indefinitely from the intermediate forms, and many more pathological and clinical observations are necessary before a useful classification of the cases can be made. In the following description the common forms are chiefly considered.

CAUSES.—The disease is more frequent in males than in females, the proportion being about three to one. It is chiefly a disease of adult life, commencing usually between twenty-five and forty-five. I have known it to begin at fourteen, and as late as seventy, and a case is recorded in which it began at twelve;* there was wasting and rigidity in the limbs, tongue, lips, and lower face. But most cases of muscular atrophy in early life are myopathic, and not spinal. Heredity is to be traced only in less than half the cases, and generally as an indirect neuropathic disposition. Rarely there is direct inheritance of the disease. Among instances that I have met with are a lady whose mother died from a similar atrophy, and another case (from which Fig. 146 is taken) in which a brother had died from some chronic disease of the cord, attended with wasting; but three cases with bulbar symptoms have been recorded, two of whom were cousins, the children of two sisters: the third, who had had syphilis, was a cousin, but his affection was untypical.† Direct inheritance seems to be most common in cases that occur late in life. When many members of a family suffer from muscular atrophy, the malady is nearly always myopathic and not spinal. The affection occurs in all classes of society, and it is doubtful whether workers with the muscles furnish a larger proportion of the cases than can be accounted for by their greater exposure to certain exciting causes.

Of these more direct causes one of the most frequent is mental distress and anxiety, and this is especially met with in females and in late life. A severe fright has been thought to excite it. Another cause is exposure to wet cold, which is also a cause of many other chronic spinal diseases. Sometimes the exposure has been habitual; sometimes a single exposure has been effective, and some neuralgic pains, indicative of the deleterious influence on the nervous system, have followed the exposure and connected it with the later wasting. The pain has been either in the part afterwards wasted, or in some other, more common, seat of neuralgia. Thus one patient, after remaining in wet clothes for twelve hours, suffered for six weeks from severe neuralgia in the left side of the face, and then the muscles of the left shoulder began to waste. Although excessive use of individual muscles may cause them to waste, it is doubtful whether this influence produces general muscular atrophy. Injury to the cord, such as results from concussion of the spine, is a rare cause. It more often produces disseminated myelitis, which may be manifested by muscular wasting combined with other symptoms. In a few instances I have known progressive atrophy, of typical characters, to slowly follow a concussion, as if this had set up a perversion of the nutrition of the nerve-elements. Still more rarely a fall, injuring one limb, has been followed by muscular atrophy commencing in this limb and becoming general. Thus a woman fell downstairs, and pitched on her left hand and wrist; she had pain in the arm for a long time; two years after the fall this arm began to waste, and the atrophy ultimately became general. The relation might be passed as an accidental coincidence, were it not that in other central diseases—even, for instance, in paralysis agitans—the symptoms may commence in an injured limb. In rare cases, the disease develops, during adult life, in a subject of old infantile palsy (polio-myelitis), and may start from the most affected part (see p. 409).

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* H. Blumenthal, "Inaug. Diss.," 'Neur. Cent.,' 1884, p. 376.

† 'Virchow's Archiv,' 1889, p. 115.

The disease sometimes succeeds syphilis, and no other cause may be traceable. As with other degenerative diseases, an interval of years elapses between the primary disease and its nervous sequel. The cases that I have seen after syphilis have been typical in course, and evidently degenerative in nature. That syphilis has some share in the causation of these cases is probable from its relation to other diseases, such as tabes; but it is noteworthy that in one case the wasting commenced during, and in another directly after, an energetic mercurial course. This relation is intelligible if the disease is due to a toxine, a result of syphilis, and not to the organisms themselves (see Tabes). Occasionally syphilis and a neurotic heredity can both be traced. In the cases in which atrophy has followed an acute specific disease, especially measles, and has begun in early life, it is probable that the malady is a chronic neuritis and not the central affection now under consideration. Such cases are known as of the "peroneal type" or "neuritic form of muscular atrophy," from the muscles in which wasting in general is first obtrusive, and are separately described at a later page. General muscular atrophy may result from lead poisoning, but this form is not, as a rule, progressive in character when its cause has ceased to act. It resembles the ordinary form of progressive muscular atrophy, however, in seat and features, and thus differs from the common atrophic palsy of the extensors that is produced by lead. In many cases of progressive muscular atrophy no cause for the disease can be traced. When obtrusive symptoms have followed some adequate cause, it is not uncommon for this to have been preceded by slight symptoms, which show that the malady already existed. A careful inquiry for such symptoms should never be neglected.

SYMPTOMS.—The definite symptoms of the disease are occasionally preceded by dull aching in the part afterwards wasted. Sometimes there is such pain in the spine or elsewhere, especially, as already stated, in the cases that are due to exposure to cold. Rarely some sensation other than pain precedes the local symptoms. Thus in one case a sense of coldness preceded the atrophy in each limb that was attacked. Weakness and wasting usually come on together, but either may first attract the attention of the patient. In the shoulder and back the loss of power is usually first noticed, and in such covered parts the wasting may become considerable before it is observed. In the hand the wasting is often first noticed, but sometimes it is the impairment of some delicate action, such as writing, that draws attention to the part. The affected muscles lose their proper shape, and there is flattening, or even a depression, where there should be a prominence. If a patient is fat, however, the wasting may cause at first very slight alteration in the external part, especially in the lower limbs.

The disease commences in the arm in nine tenths of the cases, and as frequently in one arm as in the other. It begins with almost equal frequency in the hand and in the shoulder muscles. From the part first affected the disease spreads to other parts of the same limb. Before it has attained a considerable degree in one limb, it usually shows itself in the corresponding limb on the other side; often in the muscles corresponding to those in which it commenced, sometimes to those affected second in order of time. As the muscles waste paralysis results of various character and degree, corresponding to the atrophy. In the hand the thenar muscles and interossei are usually the first to suffer. The thenar eminence becomes flattened, and the base of the first metacarpal bone becomes prominent. Of the interossei, the atrophy of the abductor indicis is especially conspicuous; the normal prominence gives place to a hollow beside the metacarpal bone when the thumb is abducted. Depressions form between the metacarpal bones on the back of the hand, and also between the flexor tendons in the palm in consequence of the wasting of the lumbricales. The forearm muscles may be next involved, the flexors usually before the extensors; and with the flexors the supinators may suffer, or they may escape until the biceps is involved. Occasionally the disease begins in the forearm, and then especially in the extensor muscles of the fingers, sometimes in those of the thumb, especially of its phalanges. The several parts of the long finger muscles may suffer unequally; the ulnar extensors usually suffer most. Of the shoulder muscles, the deltoid is generally the first to manifest the disease, and in some cases which begin in the hand the deltoid suffers before the forearm muscles. The rounded contour of the shoulder becomes changed (Figs. 7, 144, 145), and the head of the humerus can be recognised beneath the acromion. It is not rare for part of the deltoid to suffer and part escape: we have seen (p. 36) that the several portions have different functions and associations, and this probably involves a relation to separate groups of nerve-cells. The wasting of the deltoid is soon followed by that of the other muscles of the upper arm and of the scapula. The triceps usually suffers less and later than the biceps, but sometimes the reverse is the case. The supra- and infra-spinati are often affected with the deltoid. In exceptional instances the deltoid or hand muscles escape, and there are cases (perhaps a special group) in which the upper arm and shoulder muscles waste even to an extreme degree, and the forearm and hand muscles escape entirely.

In most cases, the wasting early involves the muscles of the back, and it sometimes begins in them. The middle and lower parts of the trapezius usually suffer first; the rhomboids and erectors of the spine at a later date. The affection of the trapezius is readily recognised if the patient tries to put the shoulder back (Fig. 145). The highest part of the trapezius presents a remarkable indisposition to atrophy; it often remains intact to the last, and then may contrast with the wasting below it, standing out on each side, like a cord passing from the occiput to the shoulder. Hence Duchenne termed this part the *ultimum moriens.* I have, however, seen several otherwise typical cases, (beginning either in the deltoids or in the hands,) in which the highest parts of the trapezii suffered before the middle parts. The levator anguli scapulæ also generally escapes, even when all the

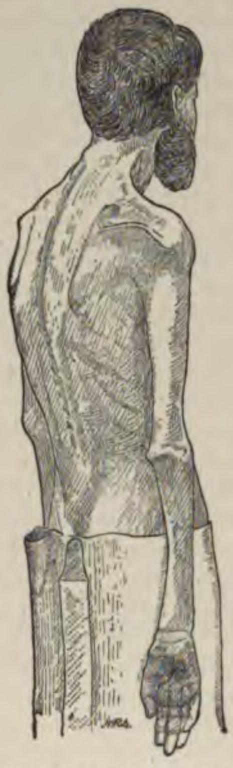

FIG. 144.—Progressive muscular atrophy. Wasting of the muscles of the back and arms; in the forearm scarcely any muscular tissue can be recognised, and in the hand all the muscular prominences have vanished. (Drawn by Dr. H. R. Spencer.)

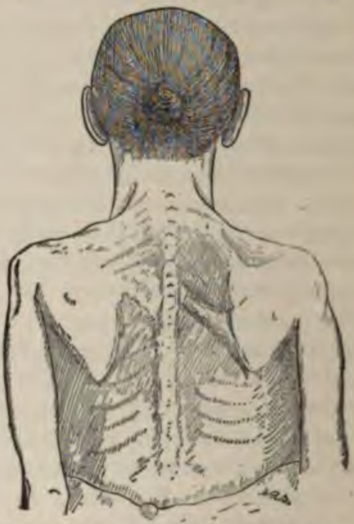

FIG. 145.—Progressive muscular atrophy. Wasting of right trapezius complete (the rhomboids remaining), of the left trapezius partial. On each side the upper part of the trapezius is wasted, and the contour of the neck is correspondingly changed. Both deltoids are also atrophied. (Drawn by Dr. Spencer.)

muscles about it are wasted. The serratus, latissimus, and pectoralis major are usually affected later. They may escape wholly or in part; isolated bundles of the pectoralis may waste, the intervening parts escaping, or either the upper or lower part of the muscle may atrophy alone. According to the affection of the muscles that are attached to the scapula, the position of the bone changes, and it becomes rotated under the influence of the muscles that are unaffected and unopposed (Fig. 7, p. 35). The muscles that extend the head on the spine often suffer in considerable degree, and from this there results a difficulty in the carriage of the head (Fig. 146). It is habitually inclined backwards, so as to balance it on the spine with but little muscular exertion; if moved forwards, it falls so that the chin touches the chest. It can be brought back into its former position only with difficulty; the patient has to incline the trunk backwards, so as to bring the head nearly into the vertical position, and then, with a sudden contraction of the sterno-mastoids, and a jerk, the head goes back into its former posture. The increased innervation of the weak extensors of the head often causes a synergic over-action of the frontalis muscles, which are normally associated with the extensors (so that the eyebrows are raised when the head is put back to look upwards.) The skin at the back of the neck lies in transverse folds when the neck is extended. The patient may be unable, when lying in bed, to move the head from side to side. The sterno-mastoids also are often wasted; either the sternal or clavicular part may be most affected. In striking contrast to the general wasting of the neck is the condition of the platysma myoides, which always escapes, and may become hypertrophied in a vain attempt at compensation.

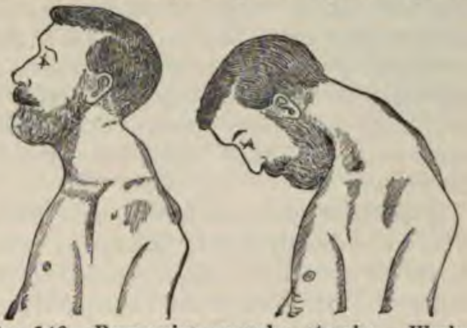

FIG 146.—Progressive muscular atrophy. Weakness of the muscles of the neck. A, habitual posture of the head, inclined backwards. B, position into which it falls if the patient attempts to keep it in the normal balance. (Drawn by Dr. Spencer.)

The muscles of respiration suffer in the majority of cases, and their impairment constitutes a grave source of danger to life. The intercostals rarely escape altogether; the diaphragm is involved in many cases, and respiration is then carried on by the intercostals and superior thoracic muscles. On the other hand, the intercostals may suffer much and the diaphragm remain free; respiration is then purely abdominal, and the walls of the thorax may be almost motionless in breathing, or there may be merely a slight movement of elevation of the upper ribs. The chest becomes flattened in front, and narrow from before back, from the influence of atmospheric pressure on the ribs, unopposed by the intercostals. Either the upper or lower intercostals may suffer most. When the diaphragm is paralysed the upper chest muscles often suffer more than the lower, and a compensatory increased movement of the lower ribs carries forward the abdominal wall, and may suggest that the diaphragm is acting when it is not. A careful examination will always prevent the error. The muscles of the abdominal wall occasionally waste, but far less frequently than those of the thorax.

Wasting in the legs is much less common than in the arms, and if it occurs is usually slighter in degree; but occasionally the disease first manifests itself in the legs, and is more intense in them than elsewhere. The glutei, extensors of the knee, and the muscles in the front and on the outer side of the lower leg, are those that are most affected. We shall presently see, however, that the legs are often paralysed when they are not wasted, and sometimes they are the seat of wasting which differs in certain features from that which is the especial characteristic of the disease.

The face almost always escapes the general atrophy, and its normal appearance may present a striking contrast to the rest of the body. In some cases, however, the lips are paralysed as part of the bulbar palsy that so often accompanies the spinal disease. A remarkable case in which the face shared the atrophy of the limbs, and the tongue escaped, has been recorded by Langer;* on the other hand, the tongue has been the first part to suffer, the arms being affected a month later.† Bulbar paralysis, however, often runs its course without muscular wasting in the limbs, and when the latter supervenes it is generally quite subsidiary. The like statement may be made of the converse relation, although it must be remembered that the anterior nerve-cells and those of the bulbar nuclei are analogous structures, and may be similarly and even simultaneously affected.

As the wasting progresses, the appearance of the parts in which it is most advanced becomes extremely changed. All trace of muscle may disappear from parts of limbs or even from an entire limb (Fig. 144); and there is usually a wasting of the adipose tissue as well as of the muscle, so that the bone seems to be covered only by fascia and skin. The transverse processes of the vertebræ may be felt in the hollow beside the spine, and the bony prominences about the shoulder may be almost as conspicuous as in a skeleton, so that beneath the acromion there may be a groove, into which the finger can be placed, in consequence of the descent of the head of the humerus from the glenoid cavity. The unequal affection of antagonistic muscles leads to various contractions and deformities. In the hand, especially, distortion is apt to occur; from preponderant paralysis of the interossei and contraction of the long flexor and extensor muscles, the "claw-like hand" develops in extreme degree (Figs. 19 and 20, p. 43). If all the muscles of a part are equally and simultaneously affected, no deformity results unless an unsupported part yields to the influence of gravitation (Fig. 144). Lordosis is very common in cases in which the trunk and hip muscles are involved. It is often the indirect result of the weakness of the extensors of the hip-joint. It is produced in the manner described in the account of pseudo-hypertrophic paralysis, in which it is almost constant.

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* Vienna Medical Society, March 17th, 1882.

† Birdsall, 'Journal of Nerv. and Ment. Dis.,' 1887, xiv, p. 256.

The electrical irritability of the wasted muscles presents changes which vary in character in different cases. When the wasting is slow, there is usually a diminution in both faradic and voltaic irritability, similar in character to each current. But the amount of contraction that can be produced is far less than normal, and progressively lessens. The irritability fails with the muscular nutrition, and when the wasting is great, only a slight contraction can be obtained, even with a strong current. When the wasting is extreme, irritability at last becomes extinct, but the voltaic irritability of the muscular fibres persists long after the faradic irritability has disappeared. It may be normal in degree, or lowered, but is seldom increased in such cases. The quality of the voltaic irritability may also be normal, but sometimes ACC (anodal closure contraction) occurs as readily, or more readily, than KCC. Tetanic contraction during the passage of the current is produced with undue readiness compared with the closure contractions, and opening contractions are often caused by currents but little stronger than those that cause closure contractions. Thus there may be the qualitative change of the reaction of degeneration without the voltaic increase (see p. 74). When there is the rapid and considerable paralysis described on p. 544, either at the onset or during the course of the disease, the palsy being followed by rapid wasting, there may be quick extinction of faradic, with actual exaltation of voltaic irritability, and the reaction of degeneration may be present in all its characteristics. Between these forms every intermediate condition may be met with, including the mixed reaction described on p. 32.

The mechanical irritability of the muscles is considerably increased; a tap causes a local contraction of the fibres struck. Moreover, spontaneous flickering contractions of parts of the muscles are very common, now of one bundle, now of another, conspicuous to the eye, although scarcely felt by the patient. This "fibrillation," as it is termed, is not invariable, nor is it confined to this disease. It may sometimes be observed in muscles that are not yet invaded by the wasting, but where it is observed, in this disease, atrophy usually follows.

In the parts affected by the characteristic wasting, all reflex action is abolished, clearly in consequence of the damage to the motor part of the reflex arc; the afferent portion of the arc is unaffected, for, as we shall see, there is no loss of sensation. The myotatic irritability there is usually only a moderate diminution in electrical excitability, which does not go on to extinction. The one condition does not seem to pass into the other; the state of rigidity and myotatic excess does not give place to muscular relaxation; atonic atrophy does not succeed the tonic wasting. In very rare cases, of which I have seen a few instances, there is a similar condition in the hands when the shoulders are the seat of simple atonic atrophy. The forearms are then rigid, moderately wasted, with myotatic excess, while the muscles of the shoulders are extremely wasted and absolutely flaccid. In still more rare cases the whole arms are thus affected, are thin and rigid, and in no part is there atonic atrophy. In one remarkable case of this character, the muscles of the trunk participated in the spasm; after coughing or yawning, respiration would be checked for a few seconds by general spasm, and whenever the patient was raised from the bed, the arms, legs, back and neck became stiff in tetanoid rigidity, the head being bent backwards.

The combinations of the ordinary atonic wasting, and loss of power without wasting and with increased myotatic irritability, are exceedingly numerous and variable. The most frequent, as already stated, is flaccid atrophy in the arms and spastic paralysis in the legs; but I have twice seen atonic atrophy below the knees, with weak but well-nourished thigh muscles and much increased knee-jerk. The variable combination of these conditions gives rise to differences that are more apparent than real, and may easily be allowed undue weight in classification. Their explanation will be considered presently.

The extreme emaciation of the most affected parts shows that the adipose tissue wastes as well as the muscles. Indeed, the sufferers usually become generally thin, often to an extreme degree. The atrophied limbs are usually cold, and may be livid or pale, but there is no tendency to acute vaso-motor disturbance in the ordinary form of the disease. I have known the skin of the face to become thin and smooth, so that, in one instance, the dark iris could be seen through the closed eyelids, but such a change is quite exceptional. In another patient there was a very remarkable form of local atrophy. Certain areas of muscles underwent wasting, the rest being normal; the affection commenced by a livid discoloration of the skin, and the wasting seemed to involve the skin, subcutaneous tissue, and muscle, causing local depressions. Considerable tracts of the trapezii were thus affected, and smaller spots in the arms and legs. The patient was a single woman of thirty-five, and the disease, which did not show a strongly progressive tendency, is probably a special affection, perhaps of the nature of scleroderma.

The functions of the sympathetic are not, as a rule, deranged. Dilatation or contraction of one pupil has been frequently observed, chiefly in association with atrophy of the muscles that are supplied from the lower part of the cervical region; and it no doubt depends on the disease of the spinal cord, and not of the sympathetic itself.

The reflex action of the iris is usually normal, and optic nerve atrophy never occurs.[*] Nystagmus is present in rare cases.

The visceral functions are usually little disturbed. Sexual power is often lost. The sphincters rarely suffer, even when the wasting is general and extreme; occasionally they are involved, and they may even suffer early. In other cases, in which the legs become weak after the wasting sets in in the arms, the affection of the sphincters may coincide with that of the legs. In the composition of the urine, slight changes have been found, but not constantly. Urea has been increased in some cases, lessened in others.[†] The quantity of lime has been found to be increased (Fromman), that of kreatin diminished, even to one tenth of the normal (Rosenthal, Langer). Glycosuria has been observed in association with bulbar symptoms.[‡] The lungs may suffer from the impairment of breathing power, when the intercostals and diaphragm are weakened.

Varieties.—The chief varieties of the disease depend on the relative distribution of the three conditions: (1) atonic atrophy, becoming extreme; (2) muscular weakness with spasm, but without wasting, or with only slight wasting; and (3) tonic atrophy, rarely extreme in degree, with myotatic excess. The commonest condition is to have atonic atrophy in the arms and upper part of the trunk, with simple weakness and spasm in the legs. Atonic atrophy in both arms and legs is less common, and the least common is tonic atrophy alone, in the arms, or universal. The last is, indeed, extremely rare. It is important to note, however, that these conditions may coexist in every degree and combination; between universal atonic atrophy on the one hand, and universal spastic paralysis without wasting on the other, there is every gradation. The latter does not come into the category now under consideration, but similar cases are met with in which there is atrophy of a few muscles (as, for instance, of the hands only), which complete the series. The varieties due to the different combinations of atrophy and paralysis have been already alluded to.

A peculiar form of muscular atrophy has been mentioned, first conspicuous in the peroneal regions * and spreading thence to other muscles of the leg and to the hands. It is characterised by occurrence in early life, and often in several members of the same family, and has been termed the "peroneal type" by Dr. H. Tooth, the "neuritic (neurotische) form" of Hoffmann. It is probably not dependent on the spinal cord, and is considered more fully at a subsequent page.

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[*] I have once met with a remarkable reflex fixation of the eyeballs in a case of advanced progressive muscular atrophy. If the patient, looking to one side, was suddenly told to look at an object on the other side, his head was instantly turned towards the second object, while the eyes remained fixed on the first, by a movement corresponding to that of the head but in the opposite direction, and then, after a few seconds, they were slowly moved towards the second object. The phenomenon continued to the end of the patient's life. As I pointed out in an account of this curious condition ('Brain,' vol. i), it is interesting evidence of a normal reflex mechanism in the fixation of the eyes; this was, as it were, isolated by disease, which lessened voluntary control over it.

[†] In a patient who weighed 7 st. 13 lbs., and whose urine varied between 760 and 960 cubic centimetres, I found the daily excretion of urea to vary between 10·7 and 15 grammes, the average being 13 grammes. This is just half the normal average for a man of that weight, which would be (according to Parkes) 24 grammes.

[‡] Rovighi and Melotti, 'Riv. Sper.,' 1888, p. 315.

Complications.—Progressive muscular atrophy is occasionally accompanied by the symptoms of some other degenerative disease of the spinal cord. The paralysis with spasm, already described, can scarcely be regarded as a complication: it is rather part of the disease, and its relation to the other symptoms will be considered when we discuss the pathology of the affection. In rare cases, muscular atrophy in the arms is accompanied by the symptoms of locomotor ataxy in the legs. By far the most frequent complication is bulbar paralysis, weakness of the lips, tongue, pharynx, and often of the laryngeal muscles. It is the expression of a degenerative process in the medulla, similar to that which, in the spinal cord, causes the affection of the limbs. It may come on at any stage of the disease, may precede the spinal symptoms, or only come on when these have attained a considerable degree. In characters, the bulbar palsy may resemble perfectly that which occurs in isolated form, and will be described among the diseases of the brain. Frequently, however, there is but little paralysis of the tongue, even when swallowing and articulation are much impaired. There may also be slight interference with articulation when there is no marked bulbar palsy. A minor complication is the neuralgic pain, which, as already stated, is sometimes troublesome in the early stage of cases that are due to cold. In several patients I have known headache to be frequent and severe throughout the course of the disease. General paralysis of the insane has been met with as an exceptional complication.†

Course.—The malady, in most cases, is steadily progressive, as its name implies, but in the rate of its progress it varies in different cases, and in the same case at different periods. It may, moreover, become stationary (sometimes as the result of treatment) at any period in its course, and when once actually arrested, it does not usually again become active. Unfortunately, the tendency to cessation is greatest in the later stages of the disease, when there is little except life to be preserved. Sometimes progress ceases at an earlier stage, and chiefly, I think, in those cases in which the atrophy is strictly symmetrical, and develops simultaneously, or almost simultaneously, on the two sides. Thus I have met with several cases in which there was symmetrical wasting in the two hands, or in certain muscles of the two forearms or of the two upper arms, and in which the atrophy, after attaining a considerable degree in its limited seat, had become stationary and continued so. Occasionally some symptom (as weakness of the sphincters) may pass away, although the atrophy progresses.

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* Cases, however, have been described more recently in which the upper limbs suffered first or simultaneously (Hoffmann, 'Deutsch. Zeitschr. f. Nervenheilk.,' 1891, i).

† Tambroni, 'Riv. Sper. de Fren., &c.,' 1887, xiii, p. 184.

When the progress at the commencement is rapid, it usually continues rapid, until the disease has attained a wide extent, although the acute local onset mentioned below may be followed by slow extension. When it begins slowly, it is usually slow throughout.

Although the disease sometimes commences in the second arm very soon after its onset, it more commonly makes some progress in its primary seat before beginning on the second side, and the interval that elapses varies with the rate of extension. It often happens that a year intervenes between the affection of the two arms, and I have known, in a very chronic case, the atrophy to slowly progress in one arm for seven years before it showed itself in the other. It is not common for the arms to be reduced to practical helplessness in less than two or three years, but the hands may become useless in as short a time as six months, while in one case the wasting, commencing at the shoulder, had invaded the whole arm in the course of a month. The shortest time in which I have known a patient to reach the last stage of the disease is nine months.

With any rate of general progress, the otherwise uniform course of the disease may be broken by the occurrence of almost sudden palsy in a certain group of muscles. Considerable loss of power, it may be absolute paralysis, comes on in a few days, or even in a few hours. It may occur at the onset of the affection. The extensors of the wrist and fingers are the muscles most commonly thus affected. The weakness is usually followed by a well-marked degenerative reaction in the muscles. I have seen several cases which began by such subacute or even acute paralysis of the extensor muscles, first in one arm and soon afterwards in the other; the initial condition closely resembled the paralysis from lead poisoning, even to the escape of the supinator longus and ext. ossis met. pol. This cause was, however, excluded, and soon the muscles of the shoulder and back presented commencing slow progressive atrophy, followed by wasting of the interossei until a typical state was reached. In another case, the quick loss of power was confined to the extensors in one arm, which were already weak and slightly wasted, most of the other muscles of the upper limbs having been long atrophied. When there is weakness of the legs without wasting, the onset of this may coincide with the atrophy of the arms, or may succeed it at any interval. In one case five years elapsed, after the arms began to waste, before the legs became weak. It is very rare for the paraplegic weakness to occur first.

The chief danger to life is from pulmonary maladies, rendered grave from the weakness of the muscles of respiration. The common complication of bulbar paralysis is another frequent cause of death, either by the interference with swallowing and nutrition, or by the laryngeal paralysis. Less commonly, death results from bedsores and septicæmia, or from intercurrent maladies.

Pathological Anatomy.—The wasting of the muscles is as evident after death as during life. They are reduced in size and pale in colour. Sometimes there is little in the tint of what remains to suggest muscular tissue. Parts of a muscle may be hardly distinguishable from adjacent fat. On the other hand, the bulk of the muscle may be dark, and pale streaks in it may mark the position of local degeneration. Under the microscope the fibres present various changes, and of these four are well defined. (1) There may be simple narrowing of the fibres, without any considerable change in their striation (Fig. 147), although the striæ often seem to be further apart than normal, and sometimes the fibrillary segmentation is unusually distinct. (2) Simple fatty degeneration, in which the transverse striation gives place to a granular appearance (Fig. 148), the granules become larger and fewer (Fig. 147) until ultimately distinct globules are scattered through the sheath. Where the muscle resembles fatty tissue to the naked eye, the microscope may show only sarcolemma sheaths containing groups of globules. (3) Muscular fibres are seen in which the sheath contains only a clear material enclosing a few fatty globules, and a few transverse striæ, faint, as if fading away. It is probable that this is not the result of fatty degeneration, but of a different process, which has been termed "vitreous degeneration"—a sort of dissolution of the striæ, indicated by the appearance of such fibres as are shown in Figs. 147, 148, 149. (4) A longitudinal striation develops in the fibre, and at first co-exists with the transverse striation; but ultimately the latter becomes indistinct, and the fibre looks like a fasciculus of longitudinal connective-tissue fibres (Fig. 150). Sometimes, with the longitudinal striation, the fibre presents a transverse striation very much finer than normal, the striæ being narrower and nearer together, as if from a division of the "sarcous elements." This change may sometimes be seen alone, and may be present in only one part of a fasciculus. Thus at one place the number of striæ in $\frac{1}{1000}$ inch was only seven, while in another region of the same fasciculus seventeen were to be counted in the same space. A tendency to transverse fissuring has also been described, but this is probably artificial. Fatty globules accumulate between the fibres (Fig. 150), accompanied in some cases with granules and masses of reddish-brown pigment. There is often also an increase of the nuclei (Fig. 148), and sometimes of the fibres of the interstitial tissue. Two or three rows of nuclei may lie between the fibres. The capillaries may be dilated and distended. It is very common to see muscular fibres that are much altered side by side with others that present a nearly normal appearance. Ultimately the sheaths become empty and shrink, and may be scarcely distinguishable from the interstitial fibrous tissue.

The peripheral nerves contain many degenerated nerve-fibres, and the terminal branches for the muscles a still larger number. If the nerves are traced up to the cord it is found that the degenerated fibres come only from the anterior roots. These are conspicuously changed to the naked eye, small and grey. They may resemble fine threads of connective tissue, and under the microscope no nerve-fibres may be found in them, or only a few may remain of normal aspect, the rest being in various stages of degeneration, or represented only by their empty sheaths. The degree of affection of the anterior roots corresponds to the wasting in the parts supplied by them. The posterior roots are normal.

FIG. 147.

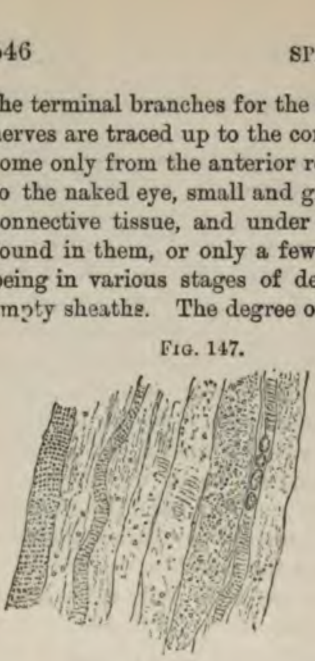
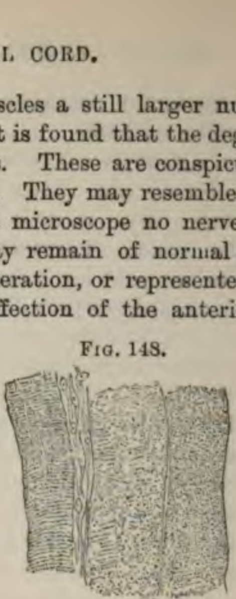


FIG. 148.



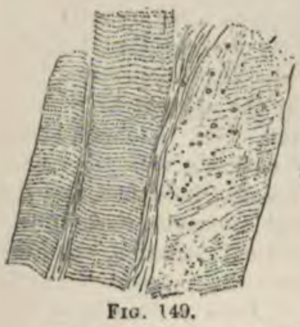


FIG. 149.

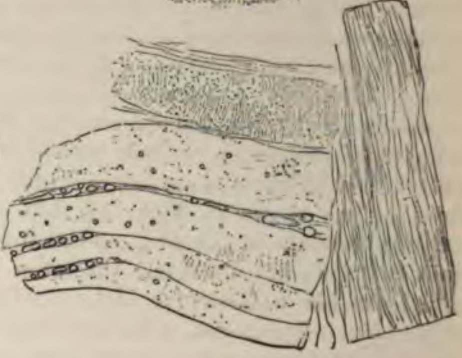


FIG. 150.

FIBRES OF WASTED MUSCLES IN PROGRESSIVE MUSCULAR ATROPHY.

FIG. 147.—Narrowed fibres with nearly normal striation; others clear, containing a few fat globules, and a few faint transverse striæ and some longitudinal striation; large globules of fat lie in the interstitial tissue in front of one narrowed fibre.

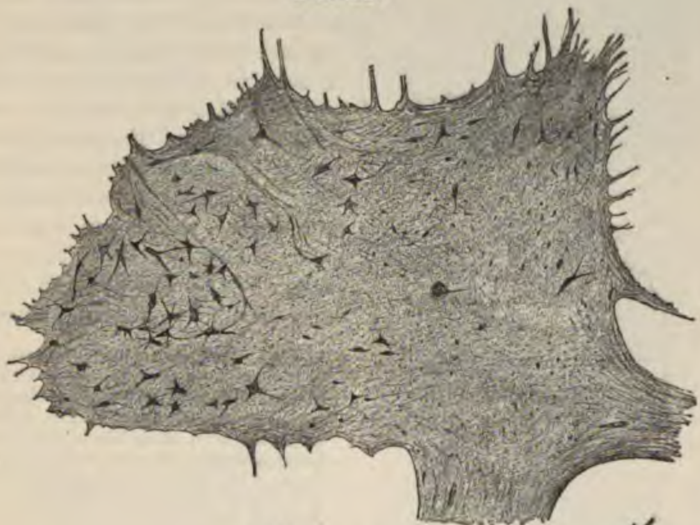
FIG. 148.—Granular degeneration: a fibre with normal transverse striation presents also indications of longitudinal striation. Increase of nuclei of interstitial tissue.

FIG. 149.—Two normal fibres and one presenting the clear homogeneous aspect, with a few fat globules and some faint striation.

FIG. 150.—Several fibres similar to that of the last figure, with globules of fat between them. On the right is a fibre which has undergone complete longitudinal striation, the normal striæ having disappeared, so that it resembles the adjacent interstitial fibrous tissue. Other fibres in the same muscle were in intermediate stages of degeneration, some transverse striation co-existing with the longitudinal striation.

The spinal cord is often softer than natural at the affected part, and the white substance of the lateral columns may be grey and translucent in aspect, especially in the cervical enlargement. Under the microscope morbid changes are seen in the anterior cornua, and also in most cases, in the antero-lateral white columns. The change in the anterior cornua corresponds in its intensity to the origin of the nerves to the most affected parts, and since the latter are usually the arms, the disease is generally most intense in the cervical enlargement. In stained sections the grey matter of the horn is less deeply tinted than normal, but in some parts it may stain more deeply, especially in the circumference of the cornu and the processes of grey matter which project into the white column. Occasionally,

FIG. 151.



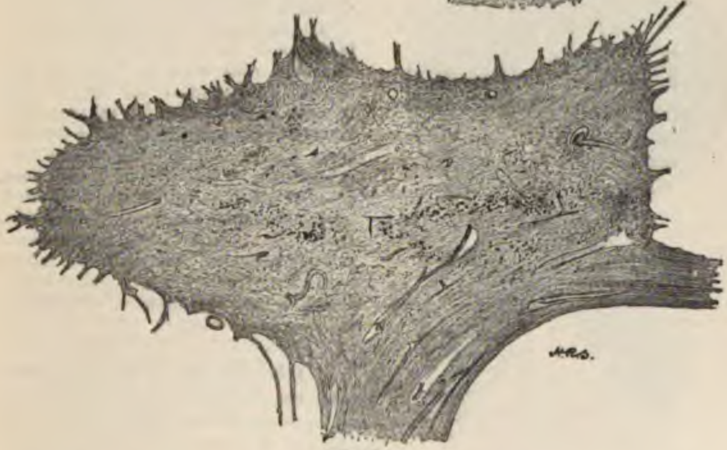
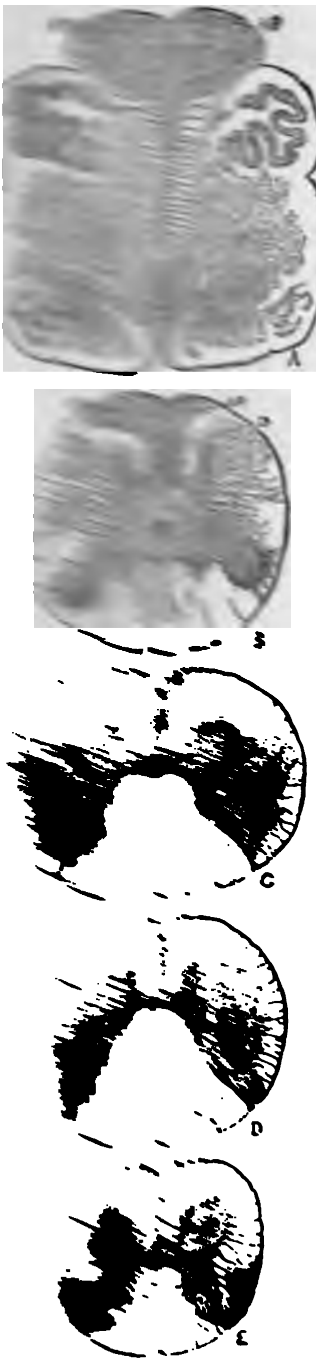


FIG. 152.

FIG. 151.—A normal anterior cornu for comparison with the next figure.
FIG. 152.—Progressive muscular atrophy; anterior cornu, cervical region.
(From drawings by Dr. H. R. Spencer.)



larger areas have a dense aspect and stain deeply, but the central part of the cornua is generally pale and wasted in aspect. Most of the large nerve-cells have disappeared; many entirely, while others are represented only by small angular bodies. Not a single large cell may be seen in a section which, in a normal cord, would contain a considerable number. Frequently, however, a few large cells can still be seen, but most of these have lost their processes and are more globular than normal. The interstitial tissue is also changed. The nerve fibrillæ waste with the cells,

FIG. 158.—Progressive muscular atrophy; degeneration of the anterior cornua and pyramidal tracts. A, medulla oblongata, complete degeneration of the anterior pyramids, A P; B, at the upper part of the decussation of the pyramids; D, the decussation of the degenerated fibres; A P, the pyramids, still incomplete. C, cervical, D, dorsal, E, lumbar sections. The degeneration of the anterior cornua is complete in C, but in E a few cells remain, for the most part without processes. In C and D the degeneration of both anterior and lateral pyramidal tracts is conspicuous; in E the anterior tract has ceased, and the lateral tract extends up to the surface of the cord, from which it is separated in C and D by the undegenerated direct cerebellar tract. In C and D the degeneration extends forwards in front of the lateral pyramidal tract, but gradually ceases. The fibres of the anterior commissure are also degenerated.[*]

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[*] In this case the arms were greatly wasted and flaccid; the legs were paralysed, rigid, with moderate wasting. The muscles of the back were also atrophied. In the arms the weakness and wasting came on at the same time, in the right arm some time before the left. The shoulder muscles suffered first, but the atrophy quickly spread, and was ultimately extreme in the muscles of the shoulders, forearms, and hands. The disease ran a rapid course; death occurred two years after the gradual onset.

and there is an increase of the small, angular, and stellate cells and other connective-tissue elements. The larger vessels are dilated and surrounded by unduly wide spaces, but there is no considerable distension of the capillaries. The cornu as a whole is not usually changed either in size or shape. Similar alterations may be traced through the dorsal region. In the lumbar enlargement the grey matter may be normal, even when it is much altered in the cervical region; but if the legs are wasted there are changes similar to those already described. When the atrophy begins in the legs, the disease may be more intense in the lumbar than in the cervical enlargement; but more often any degeneration that exists is slighter, and a larger number of normal or slightly changed cells

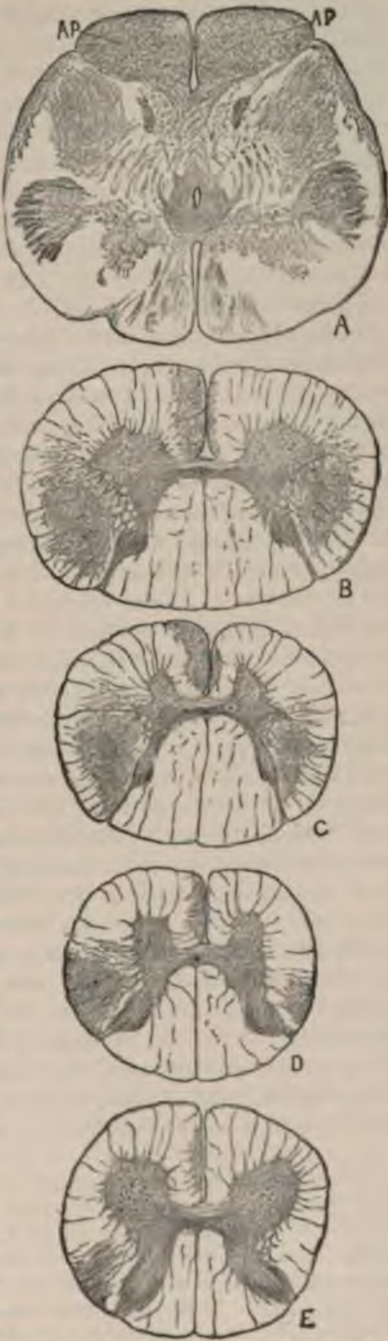

FIG. 154.—Progressive muscular atrophy. A, medulla oblongata; degeneration of the anterior pyramids, A P. B, cervical, C, first dorsal. D, first lumbar. E, mid-lumbar sections. The nerve-cells have disappeared from the anterior cornua, except in E, in which a few remain. There is complete degeneration of the pyramidal tracts, anterior and lateral. The decussation at the medulla was unequal in this case; of the left pyramid fewer fibres crossed than of the right, and hence the left anterior pyramidal tract is larger, while the right lateral tract is smaller than normal, and, moreover, has ceased at the middle of the lumbar enlargement, to which the left anterior tract extends.*

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* The patient was a man forty-eight years of age. The disease followed a severe exposure to cold. The wasting of the arms was extreme, and they were absolutely powerless. The legs were much less wasted than the arms, but very weak, the loss of power being out of proportion to the wasting.

can be seen. Sometimes certain groups of cells are little affected while others are much atrophied. The degeneration may involve to some extent the intermediate grey matter between the cornua, but here it ceases; the posterior horn is always normal.

There is distinct degeneration of the anterior root-fibres passing from the cornu through the anterior column. A few fibres may remain, but whole fasciculi appear to be replaced by fibrous tissue. There is also degeneration of the fibres of the anterior commissure, in consequence of which it stains much more deeply than normal, in the regions in which the grey matter is considerably diseased.

In the white columns there is usually considerable, and often almost complete degeneration of the pyramidal tracts, anterior and lateral.* The area of sclerosis, resulting from the degeneration of the two tracts, varies in extent according to the size of the anterior tract, and to the distance which it extends down the cord. In Fig. 154 this is illustrated in an unusual manner, in consequence of an inequality in the decussation of the pyramids at the medulla, fewer fibres of the left pyramid having crossed than of the right, so that the left half of the cord contains more than its proper share of the fibres, and the anterior tract extends into the lumbar region even further than does the lateral tract. Where the direct cerebellar tract exists, this, always unaffected, limits the sclerosis of the lateral tract on the outer side. On the inner side, the "lateral limiting layer" (see p. 221) is usually much less degenerated than the pyramidal tract, and intervenes between the latter and the grey matter; but this layer suffers in some degree, perhaps by the affection of outlying pyramidal fibres contained in it. The sclerosis does not usually cease at the anterior extremity of the pyramidal tract, but extends forward in the "mixed zone" of the lateral column, lessening in intensity, and ceasing usually opposite the outer part of the anterior cornu. It is here most intense close to the grey substance, and ceases before it reaches the surface of the cord, the antero-lateral ascending tract being unaffected. This sclerosis may be due to the degeneration of the short vertical fibres that pass between adjacent regions of the anterior cornu, and which share the degeneration of the nerve-cells. Occasionally it extends, in slighter degree, into the anterior columns. The posterior columns are always free from definite degeneration, but there may be some general increase of connective tissue throughout the whole cord. The degeneration of the pyramidal tracts is usually considerable in degree; often most of their fibres seem to have perished. If it is incomplete in the cervical region, it may be slight in the lower part of the cord, the degenerated fibres being chiefly those that end in the cervical enlargement and dorsal region. When the sclerosis is intense it is not confined to the cord; it can be traced up through the decussation (Fig. 153, B) and through the medulla oblongata, in which, as Figs. 153 and 154 show, the whole of both pyramids may be degenerated, so that they stain deeply, and no nerve-fibres can be distinguished in them. The degeneration of the pyramids has been found in several cases to extend through the pons and crus to the internal capsule, and even through the white substance to the cortex. Above the capsule, however, the pyramidal fibres are so mingled with others that their degeneration is no longer recognisable by the deeper staining of a definite tract; but it has been detected by the presence of abundant products of degeneration in the path of the fibres. In the motor cortex the large ganglion-cells have been found fewer than normal and distinctly degenerated, many having lost their processes; and, in the interstitial tissue, the spider-cells and other connective-tissue elements are increased in number.* But the degeneration of the pyramidal tracts has also been found to cease at the crus † and at the decussation.‡

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* I have not yet met with a single case of progressive muscular atrophy in which the pyramidal tracts were unaffected. Such cases are extremely rare. One has been published by Strümpell ('Zenker's Zeitsch.,' 1887, and 'Neur. Centralbl.,' 1888, p. 139) which was characterised by the general wasting of the arm, a very slow course, and the absence of leg symptoms. The spinal nerve-cells and motor nerves were degenerated. Other cases have been described by Villiers ('Journ. méd. de Bruxelles,' 1897) and by Beevor in a communication to the Neurological Society of London in 1896.

In cases in which the symptoms of bulbar paralysis were present during life, the motor nuclei of the medulla have presented changes corresponding to those of the grey matter of the spinal cord, with a similar degeneration of the nerve-cells. In other instances the degeneration of these nuclei has been slight, but in such cases the degeneration of the pyramids has been intense, and doubtless involved the fibres connecting these nuclei with the cortex of the brain.

The sympathetic nerves and ganglia, when examined, have been found normal, or have only presented such changes as are common apart from symptoms of disease of the nervous system.

PATHOLOGY.—The theories, according to which the disease was regarded as primarily one of the muscles or of the sympathetic nerves, have now only an historical interest. The constancy of the changes in the ganglion-cells of the spinal cord, the degeneration of the motor root-fibres, and the analogous effects of acute lesions of the anterior cornua, leave no doubt of the relation of the muscular wasting to the disease of the grey matter, and essentially to that of the ganglion-cells, and the fibres proceeding from them. The slowness of the change causes the impairment of the nutrition of the muscular fibres to proceed *pari passu* with that of the nerve-elements, and the interference with motor conduction to be proportioned to both. We are thus able to understand the condition of electrical excitability, and its slow

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* Koschewnikoff, 'Archives de Neurologie,' 1883, No. 18; and 'Centralbl. f. Nervenkr.,' 1885, p. 409; Marie, 'Neur. Centralbl.,' 1884, p. 61.

† Rovighi and Melotti, loc. cit.; Dornbluth, 'Neur. Centralbl.,' 1889, p. 377.

‡ Moeli, 'Arch. f. Psych.,' x, 718.

failure, as nerve and muscle degenerate together. It is only when the slow degeneration is varied by a more acute process of destruction of cells and more rapid degeneration of fibres that the muscular tissue is for a long time less damaged than the nerve-fibres, and presents paralysis in excess of the wasting, with a voltaic irritability in excess of the faradic irritability of the nerve-endings (p. 30). Thus the essential lesion of the disease is a slow decay of the lower segment of the motor path, the segment which consists of the ganglion-cells and their prolongations in the axis-cylinders of the nerve-fibres (see p. 213). To this the conspicuous symptom, the muscular wasting, is secondary. It is perhaps better thought of as a degeneration of the whole segment than as simply a lesion of the ganglion-cells, although the latter being the element on which the nutrition of the segment depends, the two views are merely different modes of stating the same fact.[*]

But the disease is rarely limited to the lower segment of the motor path. The cases mentioned in the note on p. 550 are an example of such rare limitation—an "exception that proves the rule."[†] We have seen that the pyramidal tracts are commonly degenerated, and it is probable that the degeneration often extends throughout their entire extent, and involves the motor cells of the cortex.[‡] Thus the upper segment is often degenerated as well as the lower segment. In the presence of that complete degeneration of the lower segment which causes the atonic atrophy of the muscles, the degeneration of the upper segment can cause no symptoms. The loss of power that it would produce is also caused by the degeneration of the lower segment, and the latter abolishes the myotatic irritability, excess of which is the characteristic indication of disease of the upper segment. Hence the degeneration of the upper segment of the motor path for the muscles that present the atonic atrophy cannot as a rule be recognised during life, for it cannot produce its characteristic symptoms.

What is the relation between the degeneration of the two segments, between the lateral sclerosis and the affection of the ganglion-cells? We cannot assume (as some have been inclined to do) that the affection of the upper segment is secondary to that of the lower, because a

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[*] In a few anomalous cases, degeneration of the motor cells of the cord has been met with when no degeneration could be detected in the peripheral mixed nerves (see Kronthal, 'Neur. Centralbl.,' 1891, p. 133). The interpretation of these cases is at present uncertain. Possibly complete disappearance of some fibres left the rest apparently normal, although the skill of the investigators renders this explanation difficult to accept. Many more observations, however, are needed to justify a modification of current views.

[†] Moreover the fact that these cases may not be primarily spinal must be borne in mind.

[‡] At the same time we have seen (p. 255) that the nutritional stability of nerve-fibres is less in their lower than in their upper parts, and that their isolated degeneration may be greatest in the lower parts. That this may sometimes also be the case in these pyramidal fibres is suggested by the fact already stated that the degeneration may extend no higher than the decussation of the crura cerebri.

primary lesion of the grey matter, such as occurs in polio-myelitis, does not cause ascending degeneration of the related pyramidal fibres. Moreover, even a complete interruption of the pyramidal tracts is followed by no degeneration of their upper parts. Hence the intense degeneration met with in progressive muscular atrophy cannot be regarded as secondary. Neither, when there is atonic atrophy, can we consider the affection of the upper segment to be the primary lesion, and to be the cause of that in the lower. In the seat of slight atrophy there are, as a rule, no indications of the preceding degeneration of the upper segment, which would certainly exist if this lesion were the primary change. Moreover degeneration of the upper segment does not necessarily cause any degeneration of the lower. It is a matter of every-day observation that intense degeneration of the termination of the upper segment may occur, from dorsal myelitis, for instance, without any considerable wasting of the legs, and such degeneration has never been known to excite the complete degeneration of the lower segment which causes atonic atrophy. Hence the only adequate explanation of the facts is that the degeneration of the upper and lower segments is simultaneous, or if not simultaneous, at least so far independent that neither is the cause or consequence of the other; both are the results of the same tendency to degeneration of the motor path.* Atonic muscular atrophy is thus, at least in many cases, the visible expression of a tendency to decay of the whole motor path from the cortex of the brain to the muscles.

The weakness of some parts, as the legs, with excessive myotatic irritability, often going on to spasm, is explained by the degeneration of the pyramidal fibres for the legs, the lower segment being unaffected. It is easy to understand that the affection of the upper segment, and escape of the lower, or the affection of both, may vary much in relative extent, and give rise to the multiform varieties already mentioned. In such a case the nerve-cells for the part thus paralysed are normal. This weakness of the legs usually succeeds the wasting in the arms, and in most cases in which spastic paraplegia is followed by atrophy in the arms, the latter have not shared the spastic palsy. When this condition is associated with slight wasting of the legs, without considerable change in electrical irritability, the condition exists that we have more than once considered, in which we must assume that the motor nerve-cells of the cord, while structurally intact, undergo slight changes in nutrition. In this condition there are many nerve-cells of normal appearance in the grey matter; the alterations in nutrition are too slight to cause changes in aspect, or if there are such changes we have not yet learned to detect them. They are perhaps results of the degenerative changes in the termination of the upper segment; when such degeneration is secondary to a focal lesion of the cord or brain, the changes in nutrition of the cells seldom attain such a degree as to arrest the myotatic irritability, or to cause wasting such as attends the destruction of these cells.

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* See case published by Mott Brain,' 1895.

In some cases, however, the muscular wasting may be great, although the increase of myotatic irritability persists. In these cases, as we have seen, there is considerable rigidity of the muscles throughout the whole course of their wasting, the condition that we have termed "tonic atrophy." In such a condition it is common to find that many nerve-cells have disappeared or are very small, but others remain normal or slightly changed in aspect. Apparently, in addition to the degeneration of the upper segment and to the nutritional changes just mentioned, we have then a considerable degeneration of many, but not destruction of all, the elements of the lower segment. We cannot regard this as simply secondary to the degeneration of the upper segment, for the reasons already given. It must be the expression of a distinct pathological tendency similar to that which elsewhere causes the atonic atrophy and total wasting, but slighter in extent and later in time—insufficient to prevent the less affected cells from causing rigidity under the influence of the degeneration of the upper segment. It is doubtful whether the tonic atrophy ever goes on to atonic atrophy. Theoretically conceivable, it is certain that if it ever occurs it is extremely rare. Nor does it seem that atonic atrophy ever gives place to tonic atrophy with excessive myotatic irritability. The rigidity of tonic atrophy is due to the degeneration of the upper segment, but the effect is not produced if the lower segment is already the seat of such extensive degenerative changes as abolish myotatic irritability. The pyramidal fibres for the parts that are the seat of atonic atrophy are constantly found degenerated, although the muscles have been flaccid to the last. In the rare cases (mentioned on p. 540) in which muscles with atonic atrophy become rigid towards the end of the process, it is probable, as the tenderness suggests, that the rigidity is idiopathic, due to the changes in the muscles, and is not dependent on the central nervous system. It may be the result of the increase in the interstitial connective tissue, and the longitudinal division and fibrillation by which the muscular fasciculi come to resemble bundles of connective-tissue fibres. It is also possible that similar idiopathic muscular changes may ultimately, in tonic atrophy, maintain and increase the rigidity that is primarily dependent upon the spinal cord.

It has been mentioned that the cases in which the legs present the simple palsy and spasm, or the tonic atrophy, which indicate degeneration of the pyramidal tracts, have been separated by Charcot, and termed "amyotrophic lateral sclerosis," the separation being based on the assumption that in such cases the primary lesion is the degeneration of the pyramidal tracts, and that the affection of the grey matter is secondary or "deuteropathic," even where the atrophy is atonic. We have seen also that this assumption is unwarranted so far as the atonic atrophy is concerned. It is probable that the pyramidal tracts are degenerated, if not constantly, at any rate in such a very large proportion of the cases of progressive muscular atrophy, that Charcot's distinction is, in effect, giving a new name to an old disease, and that the sequence is not that indicated by the name. Whether there are indications of lateral sclerosis or not depends on the circumstance whether the degeneration of the pyramidal fibres is or is not more extensive than the complete degeneration of the nerve-cells that causes atonic atrophy. If the latter is universal the pyramidal tracts may be totally degenerated, and yet there may be none of the characteristic indications of such degeneration. On the other hand, both arms and legs may be the seat of the spastic paralysis that indicates pyramidal degeneration, and atonic atrophy may be limited to a few muscles of the hands. Between these we have every gradation, in degree and distribution, of atonic atrophy, spastic paralysis, and tonic wasting.

The process in the grey matter has been regarded by some as a chronic inflammation. The occasional rapid increase in the symptoms may be thought to be justification for this view; but the process in general is at the degenerative extremity of the series of nerve lesions. The principle involved in this question has been already discussed, and we have seen that, whatever be the nature of the primary process, we must recognise secondary tissue changes of independent energy, and that a distinct process of inflammation may occasionally form part of these (see p. 443). The significance of the occurrence of inflammation may therefore easily be overrated, so far as concerns the question of the process, and the tissue-elements in which the disease begins.

Diagnosis.—The simultaneous and gradual onset of weakness and wasting, the slow but progressive increase and extension of the symptoms, render the diagnosis of the developed malady simple and easy. At the onset, when only a single muscle or group of muscles is affected, the question arises whether the atrophy is local or is the commencement of a wider affection. Local atrophy is said sometimes to occur from great over-use of a muscle, especially one of the small muscles of the hand; but such a cause is extremely rare, and only to be suspected on the clearest indications.

As a rule, the local atrophy from which the affection has to be distinguished is that due to disease of the nerves. The wasting from disease of single nerves or at a plexus (as the brachial) is sufficiently distinguished by its limitation, coupled with its rapid onset and associated sensory symptoms. Much more difficult is the distinction of some forms of multiple neuritis. The difficulty presents itself chiefly in two forms: (1) when the spinal affection begins as subacute atrophic palsy (see p. 544); (2) when neuritis affects chiefly motor branches, as in the arms in lead poisoning, or in the legs in alcoholism, &c. In the first case it is necessary to wait for signs of slower wasting in other parts than those first affected before a diagnosis can be made. In the second, a careful search will generally reveal other symptoms of neuritis, and a known cause is usually obtrusive.

In pachymeningitis of the cervical region, with considerable damage to the nerve-roots, the wasting in the arms may resemble that of progressive muscular atrophy, and there is often weakness with rigidity in the legs; but the wasting is less chronic in onset, and is always accompanied by distinctive sensory symptoms,—by acute pains, and usually by anæsthesia, irregular in distribution. The same distinctions suffice for the diagnosis in diseases of the nerve-roots of the cauda equina, as by a tumour. This may cause slow wasting in the legs, but there is always severe pain and loss of sensibility. Chronic disseminated myelitis may cause wide-spread muscular atrophy, but is distinguished by the presence of symptoms of irregular damage to other structures in the cord. The diagnosis from syringomyelia will be considered in the account of that disease.

From primary muscular atrophy, "idiopathic atrophy," a variety of "muscular dystrophy," as it has been termed, the diagnosis is sometimes easy, sometimes very difficult. It is easy in the pseudo-hypertrophic form, and often also in the atrophic variety (the cases in which no muscles are large), on account of its characteristic distribution, its course, the age at which it begins, and the tendency to affect many members of a family, and males more than females—features that will be presently described. Whenever several cases of muscular atrophy occur in a family, or during childhood or youth, the probability is great that they are idiopathic and not spinal. Indeed, the idiopathic form is to be suspected whenever muscular atrophy begins under twenty, unless there are distinctive spinal symptoms. But cases are sometimes met with in which idiopathic atrophy begins in adult life, and the distinction of such cases may be very difficult, and will be better understood after a perusal of the account of that disease.

PROGNOSIS.—The progressive character of the malady renders the prognosis, in every case, grave and uncertain. The chief guide is the observed tendency of the morbid process, both the fact of its actual advance and the energy it manifests. At the same time there is a possibility of arrest, greater in middle life than in old age. Some increase of atrophy in the parts already affected may occur for a short time after the process in the cord has ceased to spread—the muscular wasting going on until it corresponds to the changes in the nerves that have already taken place. The prospect of arrest seems to be greater in the cases in which the wasting is strictly symmetrical and nearly simultaneous on the two sides than in those in which it is irregular, and attacks the second side when it has attained a marked degree in the first. Spontaneous cessation unfortunately seldom tends to occur
an advanced stage is reached; but, as the result of treatment,
may take place at any stage. The danger to life is chiefly pro-
to the interference with the muscles of respiration, and to
of implication of the medulla. Bulbar symptoms

increase the gravity of the prognosis, especially when definite weakness can be recognised. Slight vague difficulty in articulation may remain stationary for years, and does not necessarily render the prognosis worse, especially if the atrophy elsewhere does not increase rapidly. If the malady ceases to advance, the prospect of any recovery depends on the rate at which the disease has progressed. Recent rapid loss of power may be to some extent recovered from, especially when the muscles present the degenerative reaction. Wasting that has existed for six months will probably persist unchanged. In a typical chronic case there is little hope of any actual recovery of tissue or power. The effects depend on a slow destruction of nerve-elements, the renewal of which seems to be impossible.

TREATMENT.—The first important element is to secure favorable conditions of life, and to maintain the general health in as perfect a state as possible. Fresh air and gentle exercise are important, but all fatiguing exertion should be avoided, and likewise all mental strain. When the patient becomes helpless, great care is necessary. Bedsores in this disease mean inattention, and may always be avoided.

Only one method of treatment has, in my own experience, shown itself capable of arresting the disease, not indeed in all, but in more than half the cases in which it has been employed.* It is the administration of strychnia by hypodermic injection. In seven almost consecutive cases, in middle life, this treatment has been followed by arrest within a month of its commencement, and the arrest has been permanent in all the cases but one. In the senile cases the treatment has failed, but in most of them the disease was in an advanced stage, and the lumbar cord had begun to suffer. In some of the cases in which the result was prompt and distinct, strychnia given by the mouth had failed. It is conceivable that the different result is due to the fact that the agent is brought into more rapid contact with the nerve-elements, perhaps in purer form. Its action may possess greater momentum, as it were, and may thus exert an influence on the nutrition of the nerve-elements much more considerable than when it is slowly absorbed from the alimentary canal. One injection daily has been given, at any convenient place. The nitrate is the most convenient salt, one fiftieth of a grain at first, quickly increased to one twentieth or one fifteenth. The injections need to be continued for months. When the malady is apparently arrested, it is well to intermit the injections for one week in three or four. Other nervine tonics seldom exert a distinct influence, but those that are useful in other degenerative diseases, as tabes, such as arsenic, may be given by the mouth at the same time as the injections are employed. In a malady so grave it is desirable to neglect nothing that may possibly exert a beneficial influence.

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* At first I regarded the apparent result with doubt, but careful and repeated observations have made it impossible to consider the arrest of the disease as other than the direct effect of the treatment. It may be asked, Why should not a larger dose by the mouth be equally effective? It does not seem to be, is the only answer that can be given.

Local treatment of the muscles has very little influence on the wasting, as may, indeed, be expected from its nature. The most sedulous and skilful use of electricity, voltaic or faradic, fails, as a rule, to produce alone any effect on the course of the disease. If the malady is progressing at the same rate in each arm, and the muscles of one arm are regularly treated with electricity, while those in the other arm are left alone, no difference can be detected in the rate of wasting on the two sides. It is possible, nevertheless, that electricity sometimes does a little good. In cases in which a rapid loss of power has occurred, and weakness is out of proportion to the wasting, some recovery is possible, and there is no doubt that the excitability of the muscular tissue is maintained for a longer time by galvanism, although the bulk of the muscle may not be influenced. In other cases all that can be said is that the influence of electricity, properly applied, is in the right direction. Moreover the disease is one of those in which patients find it hard to believe that electricity cannot help them, and the probability is that their conviction will be fostered by some medical adviser. It is one of the diseases in which unjustifiable assertions are too often made that early electrical treatment would have been successful. It may be well, therefore, if only to satisfy the patient that nothing has been left untried, that a careful course of electrical treatment should be adopted. Faradism may be used if the muscles are sensitive to it, but if they present any greater irritability to voltaism it is better to use this. It is immaterial whether the application be confined to the muscles or whether one electrode be placed over the affected part of the spinal cord. The latter method has no disadvantages, but my own observations have failed to confirm the confident statements sometimes made regarding its superiority. It is very important that the current strength employed should be moderate. Strong applications often cause much subsequent pain, and even increased disability, and should be carefully avoided. I have known a rapid increase of weakness follow a strong application in a way that convinced the patient, at least, that the two were connected.

Rubbing and massage of the muscles have been frequently employed, and of this treatment also it may be said that its influence is in the right direction, although usually inappreciable so far as the muscular atrophy is concerned. Combined with passive movement, the influence of rubbing in preventing and diminishing deformities is more distinct. No special bath treatment is of service. When the disease occurs in the subjects of syphilis, specific treatment invariably fails, and I have even known the progress of the disease to be distinctly accelerated by an energetic course of treatment both with iodide and with mercury. It is important that all treatment should be pursued in moderation, and that its effects should be carefully watched.

Arthritic Muscular Atrophy.

Articular inflammation is almost invariably attended with rapid wasting of the muscles that move the joint. This occurs equally, whatever be the cause of the inflammation, and whether this is spontaneous or traumatic. It attends chronic as well as acute inflammations, and occurs in animals if joint inflammation is produced in them.[*]

The muscles that waste are chiefly those which extend the affected joint. The atrophy is well seen in the muscles in front of the thigh when the knee is inflamed. If the ankle is affected, the calf muscles chiefly waste; if the hip, the glutei; if the wrist, the extensor muscles of the forearm; the triceps when the elbow is affected; the deltoid when the shoulder-joint is inflamed. In rheumatoid arthritis of the finger-joints such wasting is usually very conspicuous in the interossei, and especially in the abductor indicis. The atrophy, however, sometimes involves the flexors as well as the extensors, and rarely muscles of the limb that are near but do not move the affected joint. In very rare cases all the muscles of a limb have presented some wasting. Cases in which the atrophy is unusual in distribution, and especially when it is on the distal side of the affected joint, must be regarded with some suspicion, because inflammation may have spread from the joint to a nerve, and distant wasting may have been thus produced. It is said that there is sometimes, at the onset, a considerable weakening of the muscle, interfering with movement more than can be accounted for by the pain in the joint which movement causes, and that such initial palsy is transient (Valtat, Duchenne). But the pain, by its inhibitory influence, usually obscures such palsy, which is indeed confessedly rare.

The atrophy, on the other hand, may be regarded as almost constant. If the onset of the joint affection is acute, the wasting occurs rapidly; in a week or ten days a difference in the circumference of the limb may be detected by measurement. However long the affected muscle may be, the wasting involves the whole length of it, and not merely the part in the neighbourhood of the diseased joint. The degree varies; usually moderate, and sometimes slight, it is occasionally considerable, so that the femur may be readily felt when the quadriceps femoris is the seat of the wasting; or the head of the humerus may be distinct, and the acromion prominent, if the deltoid is affected. Although the whole length of the affected muscle is always involved, if the muscle is a wide and compound one some parts may suffer chiefly. Thus in the thigh all parts of the extensor may waste equally, or the rectus or vastus internus may suffer more than the other parts. The wasting increases during two or three weeks, then becomes stationary, but continues as long as the joint disease lasts. When the joint has recovered, the muscles in most cases slowly regain their normal size. Occasionally, especially when the arthritis has lasted a long time, the wasting may continue for months or years after the joint disease is at an end.

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[*] Valtat, 'Archives générales,' 1877, tome xxx. pp. 159 and 321. The subject has been discussed by Vulpian ('Leçons sur l'App. Vaso-moteur,' 1875, t. ii) and by Paget ('Lancet,' 1873, vol. ii, p. 727, in a lecture republished in 'Clinical Lectures and Essays,' 1875, p. 208).

The electrical irritability of the atrophied muscles may be normal, but is often slightly lowered, equally to faradism and voltaism. The change is trifling, to be recognised only by comparison with the other side. There is generally a distinct and sometimes a considerable local increase in myotatic irritability; the knee-jerk is excessive if the thigh muscles are affected, and a rectus-clonus can sometimes be obtained. A foot-clonus may be elicited when the ankle-joint is affected. Occasionally the increased irritability extends beyond the region of atrophy, so that, for instance, a foot-clonus may be obtained when the knee-joint and thigh muscles are affected, although there is no wasting below the knee. As an example of this, which illustrates also the occasional persistence of the atrophy, may be mentioned the case of a young man who jumped over a hoarding and twisted his left leg in doing so. He felt immediately severe pain in the knee, the joint quickly swelled, and became the seat of an acute inflammation which lasted for several weeks, and then slowly subsided. During the inflammation the thigh wasted. I saw him two years after the onset, and there was still considerable wasting, involving the whole of the extensor of the knee. The minimum circumference of the left thigh above the knee was three quarters of an inch less than that of the right, although there was no difference between the two legs below the knee. There was a slight diminution in faradic and voltaic irritability in the affected muscles. The knee-jerk was much more considerable on the left than on the right side, and there was well-marked foot-clonus in the left leg, but none in the right. A year and a half later the symptoms were unchanged. Very rarely some contracture occurs in the opponents of the atrophied muscles. Sensory symptoms are as a rule absent, but there may be slight tingling in the skin during the acute stage; it is said that areas of anæsthesia sometimes develop, but probably in such cases, inflammation has extended to a nerve in the vicinity of the joint.

PATHOLOGY.—Few facts have been ascertained regarding the condition in man. In one case, carefully investigated, the only change was in the muscles, and consisted merely in a narrowing of the fibres, one half of which were below the normal average, while no less than 84 per cent. fell short of a standard which, in health, only 21 per cent. failed to reach.* A few presented longitudinal striation (as in Fig. 150), and some of normal width were unduly tortuous; otherwise their aspect was normal. The sheath nuclei were proliferated in places, and apart from this change the intermediate substance presented local increase in quantity, especially where the fibres were narrower than normal. This observation agrees with those that have been made on animals, in which the condition has been produced, and in them, moreover, the motor nerves have been found normal (Valtat).* Indications of inflammation have been found in the joint nerves,† as might, indeed, be expected, whatever the mechanism of the atrophy. Visible changes in the spinal cord have never been discovered, but this does not exclude nutritional changes.

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* Darkschewitsch, 'Neur. Cent.,' 1891, p. 353.

We may, therefore, exclude from the possible causes, all forms of motor neuritis, both simultaneous, due to the cause of the joint inflammation, and consecutive, communicated to the adjacent nerve from the joint. The latter (a secondary extension of inflammation to the nerve) would manifestly not explain the affection of the whole of a muscle on the proximal side of the affected joint—as, for instance, atrophy of the whole extensor in arthritis of the knee—although the extension does occur, and must be recognised as an occasional event.‡ We must also recognise the possibility of a simultaneous neuritis, with acute changes in the nerve-fibres, manifested by the reaction of degeneration in the muscles. The event is, however, exceptional. It is certainly separable and to be separated from the common arthritic atrophy; but it is not easy to keep apart the two varieties of neuritis—the simultaneous and the consecutive—when a case is seen only long after the onset, because a neuritis that arises by extension may travel along the nerve and present an ultimate distribution quite different from that which it possessed at the onset.

For ordinary arthritic atrophies we must seek some other explanation. The nature of their cause long ago suggested to Paget a "reflex influence" on the muscles as the probable mechanism, and Vulpian and Charcot have suggested a similar hypothesis, assuming a derangement of the nutrition and influence of the motor cells of the cord, to be determined by the morbid impulses from the joint nerves, and to determine the alterations in the muscles. The theory receives important support from the fact that the wasting of the muscles is prevented by previous division of the posterior spinal roots (Raymond), which amounts, indeed, to an indirect demonstration of the fact that a reflex process § underlies the phenomena. It is doubtful whether we can go much further than this. By preceding hemisection of the cord it is increased on the side of the operation; but the conditions are here more complex, and the demonstration of the reflex relation less simple, than in the result of division of the roots.

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* Valtat, 'De l'Atrophie Musc., &c.,' Paris, 1877. See also Deroche, 'Étude clin. et exp.,' Paris, 1890.

† See Duplay and Cazin, 'Arch. gén.,' January, 1891.

‡ *E.g.* to the ulnar, in rheumatoid arthritis of the hand (Bury, 'Med. Chron.,' 1888, p. 182). See on this subject A. E. Garrod, 'Med.-Chir. Trans.,' lxxi, 265; also Pitres and Vaillard, 'Rev. de Méd.,' 1887, No. 6.

§ Raymond, 'Rev. de Méd.,' 1890, 374.

The increased knee-jerk, &c., that attend the wasting, and the curious fact that arthritis may set up a degeneration of the spinal cord, apparently beginning in the termination of the pyramidal fibres (see p. 493), should be kept in mind. They are certainly significant, and suggest that the influence from the joint nerves acts on the "controlling structure" of the muscle-reflex centre (see p. 240); but more facts are needed before a valid hypothesis can be framed.

Diagnosis.—The moderate degree of the wasting, with corresponding change in electrical reactions, coupled with the wide extent of the alteration, embracing the whole of the muscles involved, constitute a distinctive characteristic—distinctive, at least, when taken in connection with the preceding joint affection which is the cause of the atrophy. A primary neuritis, causing secondary arthritic adhesions, has its own features—their wider range, so far as function is concerned, and especially their limitation to nerve distribution. Hardly any other malady is likely to be confused with it, except some graver disease, while still in an early stage; but such an affection can only be distinguished by waiting until the nature of the malady has had time to show itself.

Prognosis.—When the inflammation of the joint is brief in duration, recovery of the muscles may be anticipated with confidence. In children, even after prolonged joint disease, the muscles usually regain their normal bulk. In adults the wasting often lasts for a long time after the joint is well, and a cautious prognosis should be given if the arthritis has lasted long. Even if slight wasting is persistent, normal power is usually recovered; but, as the case mentioned above shows, the symptoms sometimes continue for years. The prognosis should be especially cautious where there is a marked increase in myotatic irritability, or if the atrophy shows a disposition to involve other muscles of the limb than those concerned in moving the affected joint.

Treatment.—The chief treatment is local,—electrical stimulation of the muscles, and gentle rubbing. It is doubtful whether drugs have any influence on the condition, but small doses of strychnia may be given, or added to whatever agent is given for the joint affection. As long as the joint is inflamed, treatment rarely causes any increase in the bulk of the muscle. It is of great importance to secure the early recovery of the joint, and to avoid all influences that cause pain in it, since, as we have seen, it is apparently through the sensory nerves that the joint affection exerts its injurious influence. When the arthritis has ended, the muscles usually recover slowly without assistance, but it is probable that the local treatment accelerates the process. The form of electricity is of little consequence; either faradism or voltaism may be employed, but should only be used in sufficient strength to cause gentle contraction. The muscles act readily to faradism, and a mild faradic current is, as a rule, the best to employ. All painful stimulation of the sensory nerves should be avoided.

Muscular Atrophy from Over-use.

Muscles that are much used sometimes waste. The effect is seen most frequently in the small muscles of the hand, especially in persons of weakly constitution, who use these muscles unduly. Thus a young lady devoted many hours a day to "illuminating," during several years, and then some muscles of the thenar eminence began to waste, and became considerably atrophied. Such wasting is scarcely ever met with in the larger muscles, but has been observed in the biceps, consecutive to hypertrophy, in Sheffield smiths (Frank-Smith). The electric irritability is gradually lowered in this local wasting, equally to both currents, as it is in progressive muscular atrophy. We do not know whether this wasting from over-use is purely local, or whether the related ganglion-cells of the spinal cord fail first, and the muscular wasting is secondary to their atrophy. Gull said many years ago, "it is as reasonable to infer a lesion of the grey matter from overwork as of the muscles."* The wasting often persists when the over-use of the muscles is discontinued. Hypertrophy from over-use does not usually give place to atrophy. These two facts are somewhat in favour of the view that the lesion is primarily of the nerve-cells. The wasting in these cases shows no tendency to extension beyond its original limits. The treatment is, first, the cessation from the excessive exertion of the muscles; secondly, the improvement of the general health, and the administration of nerve tonics, especially of strychnine; and thirdly, the gentle electrical stimulation of the muscles by either faradism or voltaism.

THE MUSCULAR DYSTROPHIES.

Idiopathic atrophy, which is primarily muscular, although it is not one of the diseases of the nervous system, is commonly described with them, because it frequently presents so close a resemblance to the spinal atrophy just described as to be distinguished with some difficulty. Moreover, even the varieties that do not present this close resemblance were for a time, and indeed until lately, believed to be of central nature, to depend on the spinal cord, and to be rightly included among its diseases. Hence the custom of thus describing them has become established, and has now to be followed, although we know that it is based on a mistaken theory. At the same time, not only has the method the advantage of convenience, but it has also some measure of scientific justification. The muscles are more closely allied to the nerves than to any other structures, as regards both physiological properties and pathological susceptibility, and it is doubtful whether any other association would better befit these diseases.

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* 'Guy's Hosp. Reports,' 1862, p. 246.

The idiopathic myopathies have received much attention during the last few years, and many new facts have been ascertained with regard to the different varieties and their relations to each other. The treatise by Erb [*] has had the effect of giving unity and cohesion to what was formerly a collection of disconnected types, and has conclusively demonstrated the essential identity of the different varieties hitherto described. The so-called pseudo-hypertrophic variety of muscular affection has been recognised under this name ever since its graphic description by Duchenne.[†] The features of this were muscular weakness associated with enlargement of muscles, sometimes a few, sometimes many. He also described in the same work [‡] another form of paralysis which he supposed to be similar in its etiology to the progressive muscular atrophy of adults, and which he named "atrophie musculaire progressive de l'enfance." This was characterised by wasting of the muscles of the face as well as of the limbs, and also by its tendency to affect more than one member of a family. Leyden in 1875 [§] proposed to separate the hereditary forms of muscular atrophy from those of the Aran-Duchenne type. He pointed out that the hereditary form tends to commence in early life, to affect several members of the same family, especially the males, and he directed attention to the resemblance between the hereditary form and the pseudo-hypertrophic of Duchenne. In 1879 [‖] Gowers in a clinical lecture gave a full description, from numerous clinical observations, of pseudo-hypertrophic paralysis, and also set out all that was known of its pathological anatomy, and in this lecture clearly recognised that hypertrophy and atrophy may be combined in different proportions, and that there are cases that connect the two extremes—in some enlargement of many muscles, in others wasting in all. Such a view affords a glimpse of the group which Erb [¶] afterwards clearly outlined under the name of the "juvenile form of progressive muscular atrophy."

From a consideration of the characters and histories of these cases Erb concluded that the three varieties—the juvenile form, the hereditary form of Leyden, and pseudo-hypertrophic paralysis—were all varieties of one condition, for which he proposed the designation

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[*] 'Dystrophia muscularis progressiva,' Leipzig, 1891.

[†] 'Electrisation localisé,' 3rd edit., p. 595.

[‡] Ibid., p. 518.

[§] 'Klinik der Rück. Krank.,' ii, p. 525.

[‖] 'Lancet,' 1879.

[¶] 'Deutsch. Arch. f. klin. Med.,' Bd. xxxiv, s. 467.

"dystrophia muscularis progressiva." The next important step in the development of our knowledge took place in 1885, when Landouzy and Dejerine published* an exhaustive paper on what they named "Myopathie Atrophique." It dealt with observations of an exceedingly interesting group, the essential characteristics of which were wide-spread muscular atrophy commencing in the face, and without hypertrophy. These cases were really identical with those described by Duchenne as "atrophie musculaire progressive de l'enfance," but the later observers completed the description by the discovery from post-mortem examination that it did not depend upon, nor was it associated with, changes in the spinal cord, but that it was a muscular affection without detectable nervous lesions. They regarded their cases as constituting a distinct and separate variety of myopathy, relying upon the commencement in the face and the absence of hypertrophy, and they refused to recognise the essential identity of their cases with those of Erb, in spite of the fact that in one of their quoted cases the face was not affected, and considerable enlargement of the calves, if it was not present at the time, had been a few years before.

The latest and most important contribution to the elucidation of this disease is the work of Erb already alluded to.† In this he subjects to close examination numerous clinical records of cases of the different varieties already referred to, and makes it clear by the observation of cases of different types among members of the same family, by the description of cases which form distinct connecting links between the different varieties, and by the observation of the histological conditions in the affected muscles, that all four varieties—pseudo-hypertrophic paralysis, the juvenile form of Erb, the Landouzy-Dejerine type,—identical with the atrophie musculaire progressive de l'enfance of Duchenne—are but branches of a parent stem, and essentially the same disease. He proposes a division into two main groups, viz. (1) children, (2) adults; and in the former he would distinguish two varieties, the hypertrophic and the atrophic. In the hypertrophic he would further distinguish those with pseudo-hypertrophy and those with true hypertrophy, and in the atrophic variety those with, and those without involvement of the face. Such a division is of course an arbitrary one, for even between the two chief varieties no hard and fast line can be drawn; and the same is true in a greater degree of the minor divisions. It has, therefore, seemed best to describe the disease on the same lines as have been followed in previous editions of this book, with a due recognition of the fact that any particular case met with may partake of the characters of simple atrophy and also of pseudo-hypertrophic atrophy, and indeed may at one time be regarded as belonging to one group, and at another to the other.

All the diseases of this class seem to depend on a defective tendency in the development of the germinal tissue which forms muscles; they are essentially congenital diseases. Although they are, in most cases, merely potential maladies at the time of birth, and sometimes for years afterwards—even occasionally during a considerable part of life,—the morbid tendency does sometimes attain actual development in the earlier years. But we know nothing, or almost nothing, at present, of any other cause than this developmental tendency; we know nothing of these diseases as acquired maladies, the result of influences acting on a healthy and normal organism.

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* 'Rev. de Médecine,' 1855.

† 'Dystrophia muscularis progressiva,' Leipzig, 1891.

Another fact of their general pathology is analogous to that which we have already considered in connection with developmental diseases of the central nervous system. It depends on the double constitution of muscular as of nervous organs, on the presence of interstitial connective tissue between the contractile elements, and on the relation of both to trophic tendencies. An overgrowth of the interstitial tissue may concur with wasting of the fibres, and this overgrowth may be abnormal in its features. Hence Erb has chosen the term, already referred to, "muscular dystrophy," as a more precise designation for the class—a term which is likely to obtain general acceptance. The important fact to recognise is that there may be either a simple defect in the growth of the fibres, ultimately leading to their disappearance, or there may be, with this, an overgrowth of the connective tissue, either a simple hyperplasia, or a perverted growth in which fat-cells form. These cells increase its bulk, but are ultimately removed. The muscular fibres also sometimes present hypertrophic enlargement, but simple wasting is the most common change.

The symptoms by which these changes are manifested vary according to the nature and seat of the alteration. In all cases muscular power fails as the contractile fibres waste. The chief difference in aspect, however, depends on the seat of the affection and the effect of the interstitial changes, and especially on the frequent enlargement of muscles from the formation of fat in the fibrous tissue. This produces the semblance of hypertrophy that has led to the name "pseudo-hypertrophic paralysis" being applied to the cases in which it is present. The amount of enlargement varies much; the fibrous tissue alone may cause none, but it is sometimes great and wide-spread in the cases in which fat is formed. In all, however, a primary shrinking of the muscles, often preceded by a true enlargement of the fibres, is due to the wasting of their proper substance, the result mainly of a defective tendency of vital endurance or growth, and only in a slight degree to the influence of the interstitial tissue when this is increased in quantity.

Thus certain varieties are constituted by the external manifestation of the muscular changes. Even in the class in which pseudo-hypertrophy occurs there may be no change, or only diminution in the size of the muscles, but with increased firmness, as the accompaniment of the lessened power. When there is enlargement, this may or may not correspond in time to the loss of power, since the muscular fibres may waste either during interstitial growth, or after this tissue has undergone the atrophy which ultimately supervenes. Thus we may have two classes of cases belonging to this type, one with enlargement of muscles, few or many, the other with only wasting of muscles, the two corresponding in position and in the general course and relations of the affection.

In another form there is never an interstitial growth of fat, and probably no considerable increase of fibrous tissue; simple atrophy of the fibres is its pathological characteristic, while primary shrinking of the muscles is its external manifestation. The shoulder muscles are prone to suffer most, and in some cases there is a peculiar affection of the face. These cases are also more variable in the time of life at which the symptoms begin. But these varieties, as we have already stated, are connected by cases which to some extent combine the various features. Such combined forms forbid the separation of the types, which, nevertheless, frequently maintain so distinct a course in many members of a family, even through several generations, that we are compelled to recognise their distinctness, although we cannot divide them altogether.

The essential element in the disease has been spoken of as a "qualitative" defect, which entails an imperfect development, manifested sooner or later by the defective vitality of the proper elements of the tissue. But there is reason to believe that the defect is not always merely qualitative. In some cases the defect in certain muscles is so absolute at so early a period as to make it almost certain that these muscles or parts of muscles are congenitally absent, and that the germinal effect is thus quantitative as well as qualitative. The muscles in which this apparent failure is observed vary in the several forms, and will be mentioned in the account of these.

The peculiar form of atrophy, which differs from the others in the early affection of the muscles supplied by the peroneal nerve, is separately described, and its features are not included in these remarks.

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS (LIPOMATOUS MUSCULAR ATROPHY, &c.).

The affection thus designated usually manifests itself during the later developmental period of childhood, and clearly depends on a morbid developmental tendency, which is often present in several members of the same family. It is characterised by a progressive change in the size and diminution in the power of many muscles. The apparent cause of the change has been already mentioned.

Isolated cases, which can now be recognised as examples of this disease, were recorded in England in 1830 (Sir Charles Bell) and in

1847 (Partridge), and in Italy in 1838. A series of cases was described by Meryon in 1852, and Oppenheim in 1855, but enlargement of the muscles was not conspicuous in these.* Several remarkable examples had already come under the notice of Duchenne, who was busy exploring the field of muscular paralysis with the aid of "localised electrisation." He recognised its novel features, and published an account of it in 1861, under the name by which it has since been generally known.

ETIOLOGY.—Our knowledge of the causes of the disease is limited to a few general facts. Males furnish the majority of the cases; they suffer at least four, and perhaps seven times as frequently as females. In the latter, moreover, the malady is slighter in degree, later in development, and less frequently causes death.

The disease occurs rather less commonly in isolated cases than in family groups. The number in a family has varied from two to eight. As many as eight brothers suffered and died in the family described by Meryon, while all the daughters escaped. In a family known to the writer, four sons have suffered and none of the daughters; in another instance two daughters are affected and no sons. Thus there may be a tendency in a family to the affection of one sex, and not the other; but, on the other hand, children of both sexes may suffer in the same family. In many instances in which several members of one generation are affected, no antecedent cases can be traced in the family; the malady, while congenital, is not hereditary. In other families antecedent cases can be traced, and these are invariably on the mother's side. The disease is thus transmitted by women who are not themselves its subjects. In a case in which four brothers suffered, the mother's brother and sister were likewise affected. Again, a brother and sister were diseased, one daughter of a second sister, and three daughters of a third sister. In another instance a boy suffered, and his sister, unaffected, had two sons diseased and a daughter free, of whose children two sons were the subjects of the malady. Thus the congenital tendency is exclusively due to the maternal element in the embryo. This is also shown by a fact many times observed, that the children of the same woman, by different husbands, suffer in the same way.

Indirect hereditary tendencies, such as are indicated by the occurrence of diseases of the nervous system, can be traced so rarely that it is doubtful if they have any influence. Neither the age of

* Nevertheless Meryon's cases ('Med.-Chir. Trans.,' 1852) certainly belonged to this variety. Conclusive proof of the fact is afforded by cases in collaterals which have come under the writer's observation in near and distant branches of the same family. Other evidence of the fact is described in a 'Clinical Lecture on Pseudo-hypertrophic Paralysis' (London, Churchill, 1879). The chief literature is referred to in that lecture; the numerous papers that have appeared since have added chiefly to our knowledge of the characters of the allied forms of idiopathic atrophy, &c., and the relations of these to the pseudo-hypertrophic variety, and are referred to on a later page.

parents, nor their intemperance, appears influential, and their consanguinity becomes effective only when raised in energy by repetition. Thus, in a family known to me, the intermarriages during five generations were very numerous, and of eight children in the present family five are albinos, and two of these are the subjects of pseudo-hypertrophic paralysis. In another family of eight, whose parents were double first cousins, six children suffered from muscular dystrophy, different forms being represented in different members of the family.

The disease always manifests itself during the period of development, sometimes in the early stage of growth, at the close of infancy, often only during mid-childhood, rarely not until growth is nearly ended. In a third of the cases the first symptoms are noted when the child first attempts to walk, which is usually a little later than in healthy children; very rarely indeed the child has never walked. In about another third the child seems well until it is four, five, or six years old, and then impairment of power attracts attention. In three quarters of the cases the disease manifests itself before the tenth year. Rarely the patient is conscious of no symptoms until after puberty, at the age of eighteen or twenty; but in such apparently late onset there has been enlargement of muscles long before power became impaired, and the disease began much earlier than it seemed to do. One patient, for instance, in whom weakness was only noticed when she was twenty, had been often "chaffed," when a young girl at school, on account of her "tea-kettle calves." Neither social state nor general constitutional condition seems to influence the occurrence of the disease, but its manifestation has sometimes been apparently accelerated by influences that disturb the general health; it has been first noticed, for instance, during convalescence from some general illness.

SYMPTOMS.—Impairment of power usually attracts attention before any change is observed in the size of the muscles, or if these are noticed to be large, it is with feelings of parental pride rather than with suspicion, in spite of the fact that the children often walk clumsily, fall with ease, and rise with difficulty. The act of going upstairs is especially difficult to them; the child has to take hold of the banisters and pull himself up.

The muscles may at first present nothing unusual, especially in slight cases, or if the child is fat, as is frequently the case. But at the age of five or six years an unnatural enlargement of certain muscles is usually conspicuous, especially when there is a contrast between these and other muscles which are small. If enlargement is almost universal, it is usually great and conspicuous. The enlarged muscles usually for a time become still larger in comparison with the others, but afterwards they cease to increase and ultimately become smaller, first relatively and then absolutely. This change occurs earlier in some muscles than in others, and it may result in a condition of distinct atrophy.

in a large proportion of the cases, and with this the latissimus dorsi, which has the same action in depressing the raised arm (see p. 37). The teres major may share the wasting of the latissimus.

The other muscles of the arm suffer in diminishing degree and frequency from above downwards. The triceps and biceps are sometimes enlarged, the former more frequently than the latter, but occasionally only in one part. Both these muscles are sometimes wasted. The forearm muscles suffer in only a small minority of the cases, and the intrinsic muscles of the hand usually escape altogether.

This usual escape of the intrinsic muscles of the hand affords a very marked contrast to spinal muscular atrophy, in which they suffer early; but no rule is free from exception, and although the escape of the hands in the idiopathic affections is almost constant, it is not quite invariable; slight (or commencing) implication of the intrinsic muscles has been met with in very rare instances that were otherwise typical,[*]

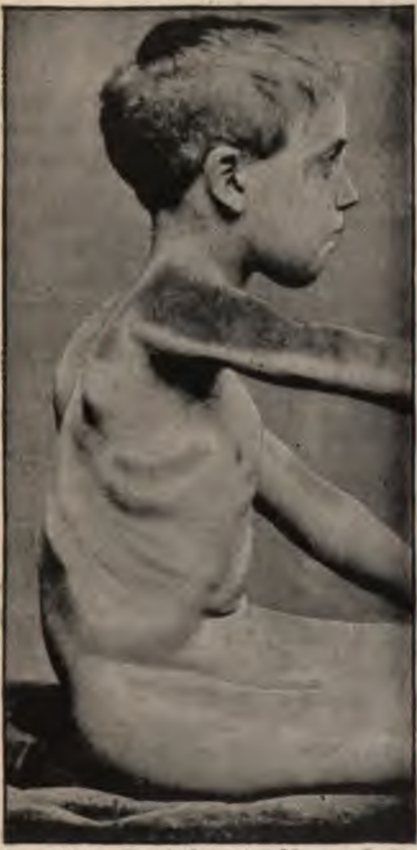

FIG. 158.—Pseudo-hypertrophic paralysis. Absence of latissimus dorsi, enlargement of infraspinatus. (From a photograph.)

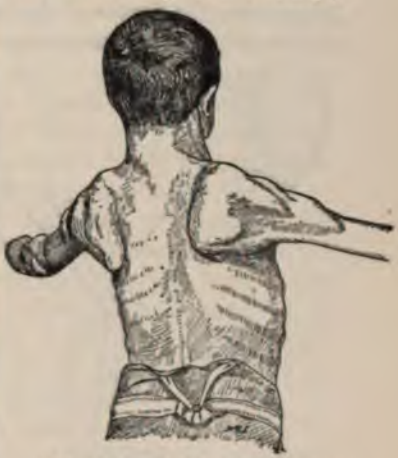

FIG. 159.—Wasting of latissimus dorsi and serratus; enlargement of infraspinatus, supraspinatus, and deltoid; atrophy of biceps and triceps. (By Dr. H. R. Spencer, from a photograph.)

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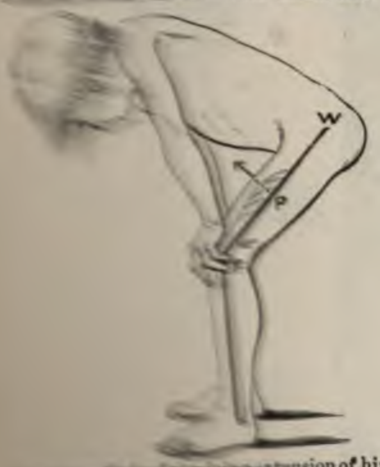
[*] *E.g.* Sachs, 'New York Neurol. Soc.,' Oct. 2nd, 1888; Baumler, 'Sudwest. Neurol. Versamml.,' Freiburg, 1888. I have once met with wasting of the extensors of both phalanges of one thumb, and enlargement of the abd. indicis has been observed (Taylor, Clin. Soc., April 24th, 1891), and fatty growth in the thenar muscles (Berger, 'Arch. f. Psych.,' Bd. xiv).

and quite distinct from the "peroneal type" to be presently mentioned.

The muscles of the neck are very seldom affected, but I have noted, in a few cases, wasting of the clavicular part of the sterno-mastoid.

Those of the face do not suffer except in extremely rare cases intermediate between this and the third type (see also Westphal, 'Charité Annalen,' 1887, xii, p. 477), but in the patient shown in Fig. 162 there was considerable enlargement of the masseters. The tongue has been increased in size in a few instances. The other muscles supplied by the cranial nerves always escape.

The diseased muscles are weak, but the impairment of power is to some extent irrespective of the change in size. The muscles that are abnormally small are generally weaker than those that are abnormally larger, and in the latter the weakness increases with the wasting. In the legs the greatest weakness is in muscles that are inaccessible to examination—the flexors of the hips; next in order of weakness come the [illegible] and the extensors of the hip. The muscles [illegible] retain considerable power for a long time, and [illegible] fail before the flexors. In the upper limbs also [illegible] are usually alone weakened during the early enlarg[illegible] subsequently the shoulder muscles suffer,



[illegible] extension of hips [illegible] paralysis. F, ful- [illegible] by the femur. P. [illegible] the power is applied [illegible] quadriceps femoris. [illegible] the ordinary mode [illegible] in which part of [illegible] by putting hands

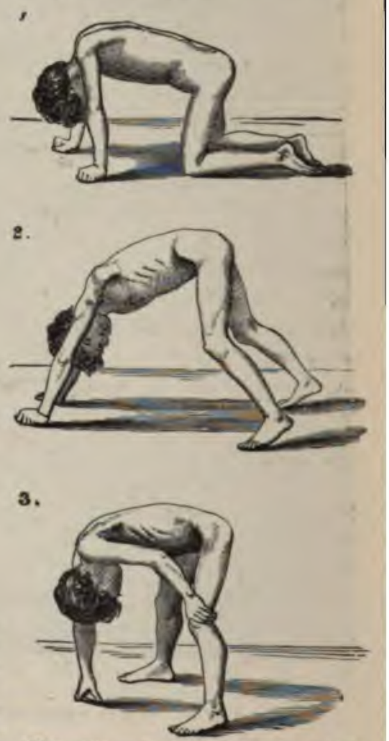


FIG. 161.—Mode of rising from the ground in pseudo-hypertrophic paralysis.

then the triceps and the biceps, while the muscles that move the hand commonly retain good power to the last.

The distribution of weakness in the legs causes certain peculiar defects of movement which are very characteristic, and some are even all but pathognomonic of the disease. The difficulty in going upstairs is especially due to the weakness of the extensors of the knee and hip. The defect of the extensors of the hip causes the gait to have a peculiar oscillating character, in which the body is so inclined as to bring the centre of gravity over each foot, on which the patient successively throws his weight, because the weak gluteus medius cannot counteract the inclination towards the leg that is off the ground unless the balance is exact. The greatest defect, however, is in the power of rising from the floor, and the most characteristic peculiarity is the mode in which this is achieved, if it be still possible, and no objects are near by which the patient can aid himself. He commonly has not sufficient power to extend the knees when the weight of the trunk is on the upper extremity of the femur, which is then a lever in which the power, applied between the fulcrum and the weight, acts at least advantage. He therefore places his hands on his knees, as in Fig. 160; and his arms thus bring much of the weight of the upper part of the trunk on the femur close to the fulcrum, between this and the power, which can then act at greater advantage. Moreover the mere weight of the head which is in front of the arms tends to aid the extension. This, indeed, may effect the extension of the knee without the aid of the extensor muscles, as any one may ascertain by observing the mobility of the patella in this attitude. When the knees are extended the power of the extensors of the hip may be sufficient to raise the body into the upright position, or the patient may aid them by an upward push with the hand as he takes it off. If, however, these extensors are weak, the hands are often moved higher and higher up the thighs, grasping alternately, and thus pushing up the trunk. To get thus the requisite support, the knees must not be quite extended; and if their extensors have no power the device cannot be employed, and the patient is altogether unable to rise. In many cases, especially when extension of the hip is easy, the patient achieves the extension of the knees in another way; he puts the hands on the ground, stretches out the legs behind him far apart, and then, the chief weight of the trunk resting on the hands, by keeping the toes on the ground and pushing the body backwards, he manages to get the knees extended, until the trunk is supported by the hands and feet, all placed as widely apart as possible (Fig. 161, 2). Next the hands are moved alternately along the ground backwards, so as to bring a larger portion of the weight of the trunk over the legs. Then one hand is placed upon the knee (Fig. 161, 3), and a push with this, and with the other hand on the ground, is sufficient to enable the extensors of the hip to bring the trunk into the upright position.

The shortening and contraction of certain muscles lead to another group of symptoms—distortions due to permanent alteration in the position of joints. Some of these are produced, as are distortions in other forms of muscular weakness, by shortening of the less affected opponents of the weaker muscles. Thus the knee-joints become fixed by the contraction of the flexors, and the elbow by the contraction of the biceps when the triceps has lost all power. These contractions only occur late, and are usually facilitated by the habitual flexion of the knee- and elbow-joints. But the deformity at the ankle-joint, which results from contraction of the calf muscles, commences earlier, before their opponents are weak, and is the result of shortening of the muscles. As a consequence of it, the patient cannot get the heels well upon the ground, and the foot cannot be flexed passively beyond a right angle. The gradual increase of the contraction results in considerable "talipes equinus;" and as power lessens, the patient is able to walk less, and the consequent loss of the extension involved in the act permits a rapid increase in the contraction. The feet, as Fig. 162 shows, soon assume a posture of extreme extension, the dorsum being in a line with the front of the leg, or forming with it a convex curve. A subluxation of the ankle-joint takes place, and the articular surface of the astragalus, its anterior extremity, and that of the os calcis, form three prominences under the skin. When this reversal of the ankle occurs, the tibialis anticus can no longer act as a flexor.

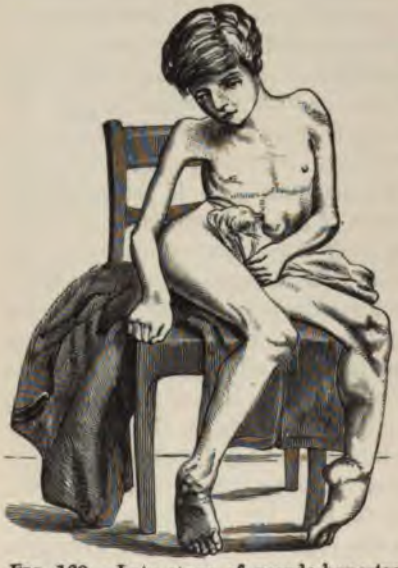


FIG. 162.—Late stage of pseudo-hypertrophic paralysis; a boy fourteen years old, with muscular contraction and wasting, and lateral curvature of the spine.

Another deformity, which is due chiefly to muscular weakness, is curvature of the spine. An antero-posterior curve, with the concavity backwards, is an early symptom of the disease, and it may become extreme, the upper part of the trunk being carried so far back that a vertical line from the scapula falls an inch or more behind the sacrum. It is due not to the weakness of the trunk muscles, but to that of the extensors of the hip, in consequence of which the pelvis is inclined forwards, carrying with it the lower lumbar vertebræ; hence the upper part of the trunk has to be held far back to keep the centre of gravity of the body over the feet. The proof of this mechanism is that when the patient sits, and the pelvis is supported on the ischial tuberosities, the lordosis disappears. It is, indeed, replaced by an opposite curve, in which the back becomes convex, clearly due to the weakness of its extensor muscles. This curve may become very great, as in the case shown in Fig. 163. The weakness of the spinal muscles also permits the occurrence of lateral curvature (Fig. 162), influenced, in its direction, by the habitual posture and the preponderance of weakness on one side or the other.

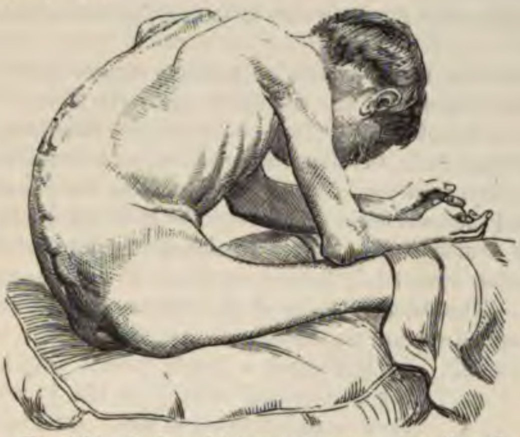


Fig. 163.—Lad aged fifteen; late stage; wasting of thighs; inability to sit upright in consequence of the weakness of the spinal muscles.

The electric irritability of the muscles is only altered when distinct weakness or wasting has set in, when it is lowered alike to faradism and voltaism. There is never any trace of degenerative reaction.

The knee-jerk may be at first normal, but as the extensors of the knee become feeble, it is always lessened and gradually disappears. It is never excessive, and in all advanced cases it is lost. Sensation is unaffected, and so also are the sphincters in the vast majority of cases Very rarely there has been, towards the end, a slight difficulty in the retension or expulsion of urine, to be regarded, perhaps, rather as a complication than as an effect of the disease. All other functions of the nervous system are commonly normal, including those of the sympathetic. The mental development of the subjects of this disease is generally beyond that of other children of the same age, doubtless on account of the indirect influence of a malady which withdraws them from active amusements; mental defect is a pure complication.

The rate of progress of the disease and its duration vary much. After some years, often between ten and fourteen, the power of standing becomes lost in consequence of the increasing weakness and the contraction of the calf muscles. When the patient ceases to walk the muscular disease makes more rapid progress, deformities become greater, and the patient may become almost helpless, except in the hands, and yet live on for several years. Death is sometimes due to [illegible], or an acute specific disease; but generally [illegible] respiratory power causes some chest affection to develop, [illegible] trifling, to become grave. Life is thus ended by [illegible] bronchitis, or by chronic lung disease—a form of [illegible] broncho-pneumonia, which develops gradually, [illegible]. There is never sufficient paralysis of [illegible] respiratory muscles to cause death directly. The duration of the [illegible] depends very much on the care which the patient [illegible].

[illegible] muscular power remains good until after [illegible]. The progress of the disease is generally slow. The patient [illegible] before motor power is much impaired. It is possi[illegible] some cases the disease never attains a considerable degree. [illegible]. However, after slight symptoms have lasted for some [illegible] a rapid increase occurs, and very few patients reach the age of [illegible]. The course of the disease is slower in girls than in boys, and [illegible] a relatively large proportion of the late cases.

[illegible]—The chief varieties of the disease depend on the age at [illegible] commencement, and on the condition of the muscles, whether they [illegible] small. In rare cases a single muscle may be large, and the rest small, as in Fig. 164, in which only the vasti were increased in size. Or everywhere and from the first the muscles are smaller than normal, and they progressively waste. Such cases are not uncommon; in the first group of cases described (by Meryon), enlargement of muscles was inconspicuous. Many cases in which all muscles are small belong properly to the form considered in the next section. But it must be remembered that there are intermediate cases which form links between the two chief types. The cases described by Meryon must be regarded as examples of the pseudo-hypertrophic form (see p. 568), but they present many points of resemblance to the "simple atrophy" described further on.

Complications.—Congenital mental weakness, due apparently to defective development of the brain, sometimes complicates pseudo-hypertrophic paralysis. In rare cases there have been indica[illegible] morbid condition of the central nervous system, [illegible] uncertain in what light the slight occasional [illegible] is to be regarded, whether as an invasion of [illegible] complication. Vigoroux has recorded [illegible] symptoms of pseudo-hypertrophic paralysis were [illegible] rigidity of Thomsen's disease. Of course [illegible] are liable, like other children, to various affections of the nervous system; I have seen both chorea and poliomyelitis as merely accidental complications.

PATHOLOGICAL ANATOMY.—It is rare at the time of death for any muscles to be actually larger than natural. Sometimes, however, they are enlarged, and the fibres themselves have been found hypertrophied, just as they have been in excised parts. There has also been found a great increase in the nuclei, atrophy of the fibres, vacuolation, and division of fibres. Most of those that are affected are below the normal size. They are pale and yellowish in colour, and often, to the naked eye, resemble perfectly masses of adipose tissue. The resemblance is not merely one of aspect. As seen under the microscope, it may be difficult for the observer to realise that he is not looking at a fatty tumour. Nothing may be at first visible but fat-cells, precisely like those of adipose tissue. Among the cells, however, are tracts of nucleated fibrous tissue, and a closer examination of these shows that the tracts contain also muscular fibres (Fig. 165), most of them much narrower than normal. They are also irregular in width; a broad fibre, for instance (as in the figure), suddenly becoming narrow.

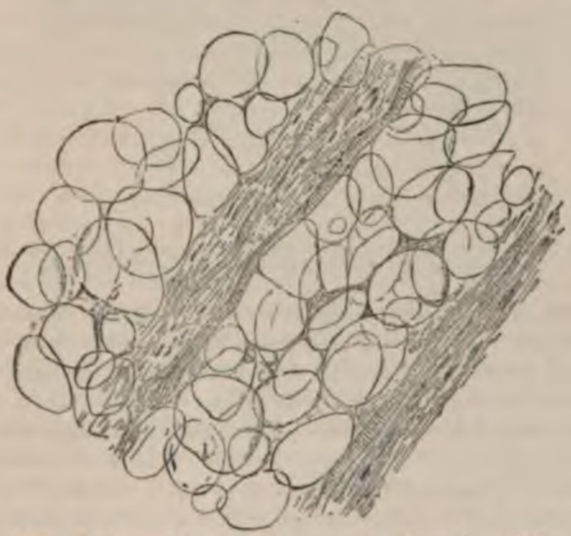


FIG. 165.—Gastrocnemius muscle; muscular fibres, irregularly narrowed and in part degenerated, lie among tracts of nucleated fibrous tissue, separated by adipose tissue.

The fibres for the most part preserve their transverse striation, but where they are narrowest this may have in part disappeared, either by granular degeneration, or, more commonly, by a simple fading of the striæ. In the narrowed fibres the striæ are sometimes farther apart than normal. In other parts broad fibres may be seen, normal or

nearly normal in aspect (Fig. 166), coursing among the fat-cells, an
accompanied by a smaller amount of fibrous tissue. Fibres occasio
ally present fatty degeneration, a longitudinal striation or fissurin
vitreous ("waxy") degeneration, or vacuolation, but these are rar
Some empty sarcolemma sheaths may be seen where the narrowing o
the fibres is greatest. In muscles that still preserve some red tin
the amount of fat is less, and there is often a relatively larger amoun
of interstitial fibrous tissue. Very rarely, in some part of a muscl
there has been only wasting of the fibres, without the interstitia
change, present elsewhere (Singer, in the triceps).

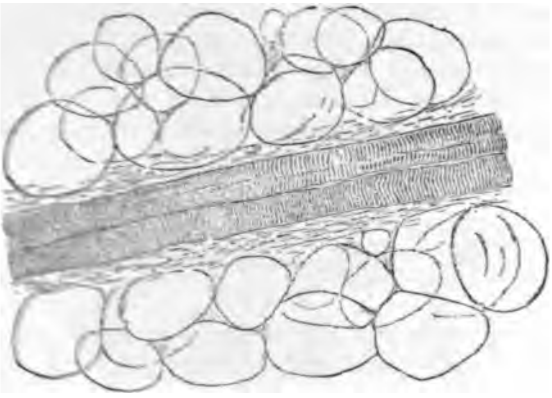


FIG. 166.—Gastrocnemius muscle; two nearly normal muscular fibres, accompanied by fibrous tissue, surrounded by fat-cells.

In some of these the interstitial tissue may be almost entirel
fibrous, a few fat-cells only being visible here and there. In suc
muscles it is common to find the fibres more damaged than i
those in which the growth is partly fatty. It is chiefly in the latte
that many normal fibres are seen. Muscular fibres larger than norm
are sometimes found after death. Such fibres have been seen in fra
ments removed during life by excision, or by a "harpoon-trocar."
has been stated that the increased size was perhaps due to a vit
contraction under the mechanical stimulus involved in the extractio
Sometimes, however, the enlargement has been unquestionable (s
note, "Muscular Hypertrophy").

The motor nerves, when examined, have been found normal. Th
condition of the sensory muscle-nerves (which terminate in t
interstitial tissue in which the primary morbid process occurs) has n
been ascertained in any instance. The spinal cord has been foun
perfectly normal in most cases in which it has been examined. In
ow there have been slight and irregular degenerative changes, as i
ne examined by Lockhart Clarke and myself;* but the anterior gre

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* 'Med.-Chir. Trans.,' vol. lvii, p. 247. In this case the cervical and dors
were normal, with the exception of here and there slight accumulations,

matter was unaffected, and the changes were probably merely associated, and not the cause of the symptoms. Hæmorrhages have been occasionally found. Probably they have occurred late in life in the degenerated tissue. The neuroglial cells have been found increased in number, and the fibres of the white substance have been found irregularly changed, in a few cases, without any constancy in the seat of the alteration.

Pathology.—The common integrity of the anterior grey matter of the cord, and especially of the motor nerve-cells, seems conclusive evidence that the disease of the muscles is not due to a primary lesion of the spinal cord. The slight irregular changes occasionally found are probably consecutive to the long inaction of the cord and deformity of the spine. Pseudo-hypertrophic paralysis is not, therefore, as was at first thought, merely a form of spinal muscular atrophy with a special muscular change. The significance of the pathological anatomy is that the malady is a primary disease of the muscles, consisting in an altered condition of the muscular fibres, leading, it may be, to enlargement and subsequently to wasting and associated overgrowth of the connective tissue, in which fat may or may not be deposited. The indication of the conditions under which the disease occurs is that it is congenital, the result of a perverted tendency of growth, inherent in the embryo, and derived from the germ from which the embryo proceeds. In this connection it is instructive to note that there is one form of congenital tumour, the structure of which is almost exactly the same as that of the muscles in pseudo-hypertrophic paralysis. Fig. 167 might be a fragment of a muscle in this disease, but it is a section of a myolipoma, the congenital character of which is emphasised by the fact that it was attached to the conus medullaris of the spinal cord of a patient whose muscles were healthy. (The tumour is shown also in Fig. 174.) It must have been due to the misplacement of some of the embryonal elements from which muscular tissue is developed, and it shows that, from such elements, the structural condition found in pseudo-hypertrophic paralysis may arise. The points of chief importance in the general pathology of the disease have been mentioned in the introductory remarks. The difficult question of the precise mechanism by which the muscular fibres suffer must be regarded as an open one. We may assume a defective vitality in them, but the conspicuous lesion is the growth of connective tissue, and by this the fibres are doubtless damaged, whether fatty tissue is formed or not. Indeed, the fibres seem to suffer more when there is

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the bottom of the fissures, of products of degeneration, probably derived from the perivascular erosion common at all ages. At the last dorsal segment, however, there was an area of granular disintegration in the intermediate grey substance on each side, in front of the posterior vesicular tract. This part was unduly translucent for half a centimetre in vertical extent, and in the middle of this area the disintegration had produced an actual cavity, across which the fibres for the cerebellar tract ran unchanged.

only fibrous tissue than when there is the fatty deposit, although it is chiefly the fat that causes the enlargement of the muscles. There seems to be a tendency to the formation of fat in the early stage of the disease, and to its removal in the later stage, since in the former the muscles may often be observed to increase in size, and in the latter they become smaller, partly from some removal of fat and partly from the increasing atrophy of the muscular fibres. It is then that the greatest impairment of power occurs. In the cases in which the muscles are small from the first, the tendency to the deposit of fat seems slight, and the condition is an almost pure muscular sclerosis. The late shortening which occurs seems to be due to the contraction of the interstitial fibrous tissue, and it often coincides with the shrinkage from the removal of some of the fat, and the atrophy of the muscular fibres.

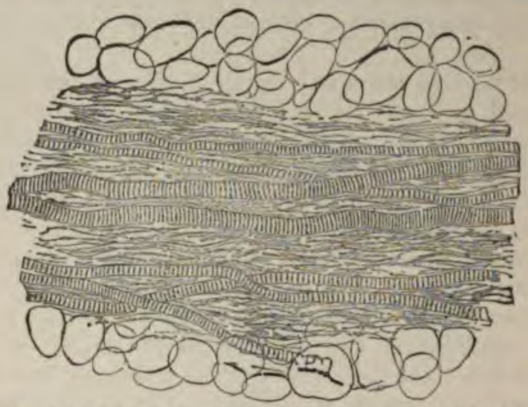

Fig. 167.—Section of a myolipoma which was attached to the spinal cord of a man suffering from locomotor ataxy, for comparison with Figs. 165 and 166.

If it be true that the latissimus dorsi and lower half of the pectoralis are sometimes congenitally absent in pseudo-hypertrophic paralysis, the fact is quite consistent with the explanation of the nature of the disease given above. These two muscles stand perhaps lowest, in functional importance, among the muscles of the body, being used chiefly for the rare depression of the arm against a resistance (see p. 37). It is readily intelligible that a defect in the embryonal tissue of the muscular system should be quantitative as well as qualitative.

The loss of the knee-jerk is sufficiently explained by the lesion of the muscles. According to the theory that the irritability on which the jerk depends is due to a muscle-reflex action, the loss is readily intelligible, since the afferent influence is due to the stimulation, by tension, of the sensory muscle-nerves, and these end (or begin) in the interstitial tissue, which is the seat of the primary morbid process in this disease. But the wasting of the muscular fibres must also be capable of abolishing the knee-jerk when the atrophy reaches a considerable degree, and it is also possible that the motor nerve-endings share this structural damage. On any theory of the nature of the knee-jerk, this will explain its loss. It is only when the muscular changes have attained a considerable degree that the loss occurs. The fact therefore does not, in itself, suggest any lesion of the spinal cord. If the malady is thus one of a morbid developmental tendency, it is not surprising that a tendency to analogous developmental diseases should be sometimes present in the nervous system, and that thus imbecility and epilepsy on the one hand, or albinism on the other, should be occasionally associated with the disease.

DIAGNOSIS.—The diagnosis of the disease is usually easy if its characters are known. The peculiarity of gait and the mode of rising from the floor, the age of the patient, and the progressive character of the impairment, are in themselves sufficient to suggest the affection, and examination reveals enlargement and often contraction of the calf muscles, and a change in the size of others, which confirm the diagnosis. The mode of rising is not absolutely pathognomonic, and it has misled even good observers; it is occasionally acquired in other diseases in which there is a gradual weakening of the extensors of the hip and knee. It is met with equally in the simple idiopathic atrophy, but is so rare from any other cause as to be of very great diagnostic suggestiveness.

Of the condition of the muscles, that which is most characteristic is the combination of enlargement of the infraspinatus with a wasting of the latissimus and lower part of the pectoralis. I pointed out some years ago * that this condition, which is seldom absent, is of very high diagnostic importance, and subsequent observations have fully confirmed the opinion. Next in importance is the enlargement of the calf muscles, especially in combination with contracture that cannot be overcome. In general, the enlargement and diminution of neighbouring muscles is very significant, but it must be remembered that they may be gravely diseased, and yet of normal size, or from the first smaller than normal. In such cases they are often hard, and the distribution of the affection is the same as that of the double change in typical cases. Such cases illustrate the relation of this form to the simple atrophy described in the next section.

The disease with which confusion is most common is the so-called "congenital spastic paraplegia" (p. 495). Both diseases affect children; in both there are weakness of the legs and contraction of the calf muscles, and in both the muscles are frequently large. The chief distinctions have been already mentioned. The most important are the preservation and excess of the knee-jerk in spastic paraplegia, the tendency to spasm of the legs—the facts that the contracture is active and can be overcome, that the patient does not rise from the ground

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* 'Pseudo-hypertrophic Muscular Paralysis,' London, 1879.

in the way peculiar to pseudo-hypertrophic paralysis,—and the opposite tendency of the two diseases. Congenital dislocation of both hips sometimes presents a superficial resemblance. Spinal muscular atrophy is only likely to be confused with the simple atrophy.

Between the two forms of idiopathic atrophy the distinction is scarcely one of diagnosis proper; it is rather a question of the category in which a case should be placed. The most important distinction is the freedom of the calf muscles from enlargement in the atrophic form. The face is not affected in pseudo-hypertrophy as it often is in idiopathic atrophy, except only in some intermediate instances. If more than one member of a family is affected, some of the sufferers will usually present characteristic symptoms of the special form, for it is remarkable with what constancy the two types generally remain distinct (see p. 567).

Prognosis.—In the case of any child with pseudo-hypertrophic paralysis, the prognosis is most grave. It is almost certain that each year will bring increasing disability, and that the patient will not reach adult life. It is only when the disease develops late, and the symptoms do not become considerable until after twenty years of age, that there is a possibility that the disease may not attain its ultimate degree, but even in such cases this hope is seldom realised. In any case, and at any age, it is unlikely that the patient will live more than seven years after the power of standing is lost. But even in this condition arrest seems to take place in the progress of the disease. One patient is known to the writer who presents a typical picture of the pseudo-hypertrophic type, who is now over forty, and has never been able to run. He has remained stationary certainly during the last eight years, probably longer.

Treatment.—As a congenital developmental malady, pseudo-hypertrophic paralysis is one of those diseases in which medicine is necessarily powerless to cope with the essential elements of the process. As may be therefore expected, no drug has been found to exert an influence on the course of the affection, although such nervine tonics as phosphorus and arsenic have been thought sometimes to retard for a time the progress of the weakness. The stimulation of the muscles by electricity has been employed and advocated, but, however sedulously employed, no distinct result follows the use of either faradism or voltaism. It must be remembered, moreover, that electricity is a very feeble agent in stimulating the growth of muscular fibres, compared with the physiological stimulus of voluntary effort. Muscular exercise may reasonably be looked to in order to make up, in some degree, what is lacking in this disease, and does seem to have some influence in retarding the failure of power. It may perhaps induce further growth, or greater power in the muscular fibres that have not yet suffered, or actually supplement the defective trophic energy. When muscular exercise is stopped, there is certainly a quicker failure of strength. Hence it is desirable that the patient should carry out carefully planned gymnastic exercises, so arranged as to call into action the muscles that most need help. These, thoroughly persevered in, have seemed, more than any other means, to retard the disease. Although they have not in any case arrested it, the trouble that is necessary to arrange the method is certainly well spent. Rubbing and massage improve the circulation, and help, especially when combined with passive movements, to lessen the tendency to muscular contraction and consequent deformities.

The influence of muscular exercise renders it very important to maintain locomotion as long as possible. The ability to stand and walk is generally lost, through the contraction of the calf muscles, some time before the muscular weakness would take the patient off his feet. In such cases tenotomy may restore the power of walking for some years, and when the contracture returns, its removal has, a second time, enabled walking to be resumed. The operation is thus distinctly beneficial, and should be performed as soon as the actual need for it arises, and division of the tendon is far better than any imperfect substitute.

During the later stages of the disease great care is required to preserve the patient from catarrh, which helps to excite the pulmonary mischief that so often ends life. Similar care is also needed during any intercurrent malady which the patient may contract.

Simple Idiopathic Muscular Atrophy.*

The cases in which there is no muscular enlargement, in which wasting is manifest from the first in the size of the muscles, are much more rare than the pseudo-hypertrophic disease. They belong to several types, more or less distinct, of which the most common is that to which Erb has given the name of "juvenile," and of which most instances are probably the pseudo-hypertrophic disease without muscular enlargement; others are more special, since the lower limb muscles have suffered but little. A more striking variety is that which is characterised by the affection of the muscles of the face in addition to those of the shoulder girdles—the "facio-scapulo-humeral type" of

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* The most important writings on the subject (besides those specially quoted) are those of Duchenne in 'Electrisation localisé' (p. 60 of Poore's translation, published by the New Sydenham Society); Barsickow, 'Inaug. Dissert.,' Halle, 1872; Leyden, 'Klin. d. Ruckenm. Krank.,' Bd. ii, p. 525; Möbius, "Hered. Nervenk.," Volkmann's 'Klin. Vorträge,' No. 171; Landouzy and Dejerine, 'Revue de Méd.,' 1885, pp. 81 and 251; Marie and Guinon (a series of cases observed at the Salpêtrière), ib., 1885; Sachs, 'New York Med. Journ.,' Dec. 15th, 1888; Hitzig, 'Berlin. klin. Wochenschr.,' 1888; Singer, 'Zeitsch. f. Heilk.,' Bd. viii. Especially valuable are the papers by Erb, 'Deut. Archiv f. klin. Med.,' Bd. xxxiv, 1884, and 'Neurol. Centralbl.,' July 1st, 1886, with numerous later writings. Bibliographical references, &c., will be found also in Tooth's 'Thesis on the Peroneal Type,' and in my Lecture on Pseudo-hypertrophic Paralysis (1879), and in Sachs' article (loc. cit.).

Landouzy and Dejerine. In a third variety the affection begins in the legs, and is peculiarly slow in course. Some other types may ultimately be differentiated, but it is important to recognise the fact that even those that we can now distinguish, as the most pronounced in their features, do not keep entirely distinct. They are connected, by intermediate forms, not only with each other, but even with the pseudo-hypertrophic disease, as already mentioned.* But these are unusual; as a rule the two latter types remain distinct in the families in which they occur, and this is the justification for their separate description. In those families in which simple muscular atrophy occurs, however numerous the cases, however different the distribution of the disease, and however various the ages at which it begins, cases rarely present the distinctive characters of pseudo-hypertrophic paralysis.

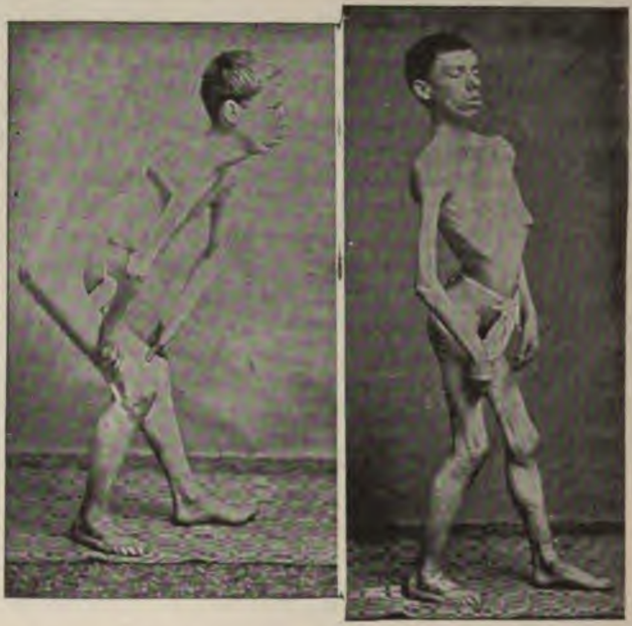

FIG. 168.—Simple idiopathic muscular atrophy. From photographs kindly lent by Dr. J. H. Crocker. Particulars of these cases and many similar are given in an excellent thesis on this disease by Dr. Crocker.

There is less constant separation between the simple "juvenile" and the facial forms. Although they often keep distinct, the affection of the face has been absent in a few cases in a family in which it was the first part to be affected in most sufferers. Thus a man with simple atrophy, beginning at fourteen in the shoulder and thigh muscles, whose face was unaffected, had a daughter who began to suffer at eleven, and in whom the face, scapular, and arm muscles were involved.* The peculiar "peroneal type" is not included in this outline, and the statements here made do not apply to it.

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* *E. g.* case described and collected by Marie and Guinon (loc. cit.), connecting the pseudo-hypertrophic form with both the juvenile and the facial.

CAUSES.—We are able to trace no cause beyond the congenital tendency, already considered, shown by the occurrence of many cases in the same family, and in more than one generation. In one remarkable series recorded by Barsickow, twenty-four cases were distributed through five generations, and the disease was also traced through five generations in a group described by Landouzy and Dejerine It is very rare for the disease to be confined to one generation, far more rare than for pseudo-hypertrophic paralysis to be so confined. But, as in the case of most congenital hereditary diseases, cases that are apparently isolated are occasionally met with. Such isolated cases are rare—more so, probably, than in the pseudo-hypertrophic form; although wider observation may show that they are more frequent than we now suspect. In the families of the patients shown in Figs. 169–172 no example of analogous disease could be heard of.

Both sexes suffer; in a few families, females chiefly; in others, males; in most, both have been affected. The age at which the disease first manifests itself is extremely variable. It may begin as early as two † or three,‡ and as late as sixty years. But the onset is seldom during childhood; in the majority of cases the disease shows itself between fifteen and thirty-five; that is, during the later period of growth and the early period of adult life. Even in the same family, the variations may be extreme; in that described by Barsickow the date of the onset of seventeen cases was known, and was as follows:—in one at 12; in four between 15 and 20; in seven between 20 and 30; in three between 30 and 40; in two after 40. A woman aged fifty-two began to suffer at 30, but her son at 3 years of age.§ Sex has no influence on the date of onset, nor, as a rule, can any relation be traced between the date and the place at which the wasting begins. When the wasting begins in the face, the disease more frequently commences in childhood than when the first symptoms are in the limbs, but in some instances the atrophy has commenced in the face late in life, and this in the same family in which other sufferers have been young. Thus in the sexual proclivity, and in the date of onset, there is a marked difference between this form and pseudo-hypertrophy, the latter showing a stronger tendency to affect males, and to manifest itself in childhood. The "juvenile" form, as a rule, presents less difference.

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* Troisier and Guinon, 'Rev. de Méd.,' 1889, p. 48. See also Singer, 'Zeit. f Heilk.,' viii, p. 229.

† Kreske, 'Münch. med. Wochenschr.,' 1886.

‡ Landouzy and Dejerine, loc. cit.

§ Ibid.

As a rule, no direct exciting cause can be traced. In a few instances the onset has succeeded some other morbid process, such as chlorosis, acute disease, or rheumatic affections due to exposure to cold; and the depression of general health resulting from these may have determined the time of onset, but is not likely to have done more. In other individuals of the same families the disease has developed without the aid of any exciting influence.

SYMPTOMS.—The onset is always gradual. Weakness and wasting come on together, and are noticed simultaneously, unless the commencing atrophy is concealed by subcutaneous fat. The atrophy generally begins in the upper arm and shoulder muscles, having, in the most common "juvenile" form, a similar distribution in these parts to that of pseudo-hypertrophic paralysis. In the facial form, however, this part usually suffers first, as in the lad figured on p. 589. In rare cases the wasting not only begins in the legs, but remains limited or almost limited to them. In the part first affected the disease slowly increases, and thence it usually spreads. The onset may be symmetrical on the two sides, or one side may suffer some time before the other.

Of the arm muscles, the weakness and wasting are noticed first in the biceps and triceps, and with these the supinator longus often suffers. But examination generally shows that the lower part of the pectoralis and latissimus dorsi are greatly wasted, a loss of which the patient may be little aware, on account of the relative unimportance of these muscles. Sometimes the upper part of the pectoralis, and even the pectoralis minor, are also affected. The tendency to atrophy of the lower part of the pectoralis and latissimus is a character which, as already stated, is common to this disease and pseudo-hypertrophic paralysis, but not invariable in either. The deltoids are rarely involved; in many cases they are normal; sometimes they have been thought to be unduly large; in a few instances they have been wasted. The serratus magnus is often affected (Fig. 172), but may escape, even in a severe case. The supraspinatus and infraspinatus may also suffer, but are often normal, or even enlarged; the trapezius and rhomboids have been affected in many cases; sometimes much atrophied, especially in the "juvenile" form.

The forearm muscles generally escape, with the exception of the supinator longus. Occasionally there has been some weakness of the long extensors or flexors of the fingers, with or without slight visible wasting. In the case mentioned on the next page the extensors of the phalanges of the thumb were involved on the left, and the radial extensor of the wrist on the right side. Rarely the forearm muscles have been much atrophied. In several instances there has been some atrophy of the small muscles of the hands, the thenar and interosseal muscles, or the interossei only, as in the case of Landouzy and Dejerine; but in the majority the integrity of these muscles is a marked feature of the disease and a contrast to the spinal form.

The affection of the face is peculiar. There is commonly a failure of the zygomatic muscles, and, in consequence, a loss of the naso-labial furrow, and a curious alteration in the smile; instead of the angles of the mouth being drawn outwards and upwards, they are moved upwards by the elevators of the upper lip and angle of the mouth. The orbicularis oris is also affected, and, in consequence, the lips are habitually separated, the lower lip projects, the patient cannot "pout" or whistle, and the articulation of labials is imperfect. The face has a dull expression, and the aspect is very peculiar; it has been termed, by Landouzy and Dejerine, the "myopathic face." In one case, observed by them, the face was unequally involved on the two sides. Rarely (as in Fig. 171) the frontales have been involved, and the orbicularis palpebrarum has been weak, and the eyes cannot be completely closed. Weakness of the eyelids was supposed to be the cause of distinct exophthalmos present in one woman.[*] In the case described by Kreske, inability to close the eyes was noted at three, and at ten the paralysis of the face was absolute. Wasting of the muscles may be indistinct, because the contour of the face is only to a slight degree influenced by the substance of the muscles The projecting lower lip may, indeed, appear to be thicker than normal. In many cases the buccinators have been affected, in some instances they have been normal, and then have drawn out the angles of the mouth in smiling. The tongue has been always unaffected, and so also have the pharynx, larynx, muscles of mastication, and the eyeball muscles [†]

The muscles of the spine have sometimes been normal, sometimes they have been considerably atrophied.[‡] The intercostals are often affected in the later stages, but rarely in extreme degree. The diaphragm also sometimes suffers. The abdominal muscles have been involved in only a few instances. In the legs the muscles most commonly affected are the flexors of the hip, the extensors of the knee, and less frequently the glutei. The muscles below the knee have escaped in many cases; when they have suffered, the atrophy has often been general. When the peroneal and anterior tibial muscles have been specially affected, as in the case shown in Fig. 172, the case has generally been of the peculiar "peroneal" type to be presently mentioned, although it is possible that some instances of the kind are on the borders of the group now under consideration.

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[*] Landouzy and Dejerine.

[†] In a singular case under my care some years ago, an affection of the facial muscles, similar to that of idiopathic muscular atrophy, was associated with paralysis of ocular muscles. The patient was a girl twenty-seven years of age; there were no indications of syphilis, nor could any history of muscular atrophy in the family be ascertained. The ocular palsy commenced gradually at twenty-four, and increased until the movements of both eyes upwards, of the left eye inwards, and the right outwards, were lost, and all other movements were weakened. The eyelids drooped slightly; the internal ocular muscles were normal. The affection of the face followed that of the eyes; the zygomatic muscles were powerless, so that the smile consisted only in elevation of the upper lip; the orbicularis was weak. The palate, pharynx, and larynx were normal. The arms became feeble, and the flexors of the hips almost powerless. There was no visible change in the nutrition or electrical irritability of the muscles; the knee-jerk was normal. If the case was central, as it appeared to be, the peculiar affection of the lips and zygomatics is not confined to idiopathic muscular atrophy. If the disease was muscular, the eyeball muscles do not invariably escape. With this case may be noted one recorded by Oppenheim ('Charité Annalen,' xiii) as the "juvenile" form, with derangement of the lateral movement of the eyes, nystagmus, and some laryngeal palsy. The shoulder and thigh muscles suffered chiefly, but below the knee, the peronei—a noteworthy aberration from the type.

[‡] Atrophy began in the back muscles at forty-four in a case recorded by Musso

The electric irritability of the affected muscles is usually lessened in proportion to the wasting, and equally to both currents. Indeed, the diminution seems sometimes out of proportion to the wasting, and great when the atrophy is slight. There is no trace of degenerative reaction, and there is not even the longer persistence of voltaic than of faradic irritability which occurs in the more chronic cases of spinal atrophy. Fibrillation is almost always absent, but not quite invariably; hence its absence cannot be made distinctive, still less its presence. Myotatic irritability is lessened or lost; it is never increased. Some shortening of muscles has been occasionally noted, frequently in the calf muscles, rarely in the biceps.

All other functions of the nervous system are unaffected. Sensibility is normal. In a few cases transient rheumatoid pains have accompanied a rapid development of the disease in the arms, but in the vast majority of cases the disease is painless. The sphincters are unaffected, and there is no tendency to trophic or vaso-motor disturbance.

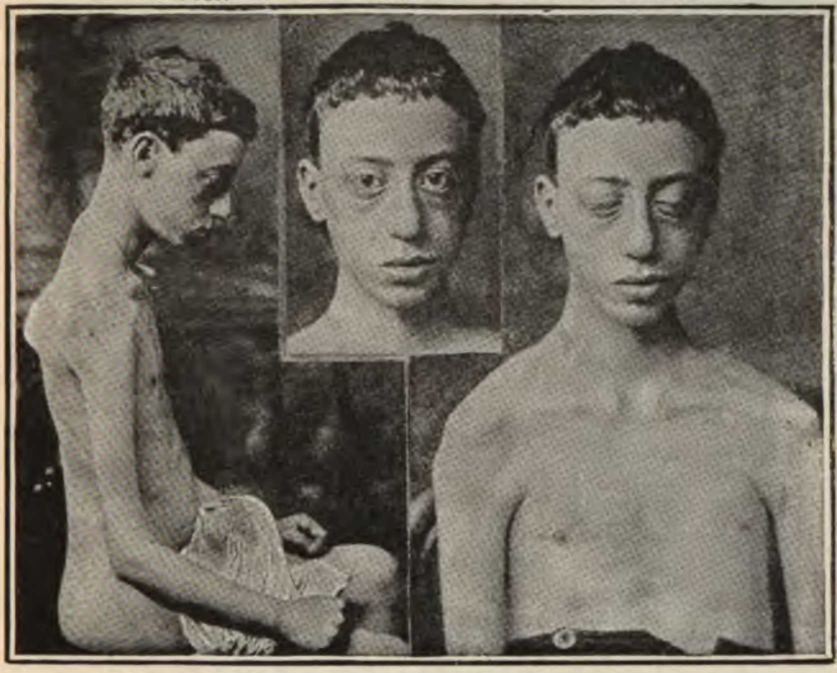
As the muscular atrophy progresses, the form of the affected parts becomes changed just as in spinal atrophy. Deformities may also occur, chiefly from the shortening of less affected muscles, but these rarely reach the considerable degree common in other forms of muscular wasting. Lordosis occurs in the upright posture, and is probably due to the same mechanism as in pseudo-hypertrophic paralysis, ceasing, as in that disease, when the patient sits (compare Figs. 169 and 172). When the muscles of the lower leg are involved talipes may develop.

The course and duration of the disease are exceedingly variable. The atrophy may remain limited to the part in which it begins. The face alone has been affected in some members of a family, although in other members the limbs subsequently suffered. In cases in which the wasting spreads, years may intervene before the extension takes place. In one case, for instance, the right arm became affected at

('Riv. Clin.,' June, 1887), and twelve years later had involved also the legs. The patient's mother, two brothers, sister, and maternal uncle suffered in the same way at the same age.

nineteen, the left at twenty-five, and the legs at thirty. In another the arms began to waste at thirty-five, power of standing was only lost at fifty-five, and the patient lived till seventy-five. Even when the malady

FIG. 169. FIG. 170. FIG. 171.



FIGS. 169 TO 171.—Idiopathic muscular atrophy affecting the face. Fig. 169 shows the wasting of the deltoid upper arm muscles and supinator longus (while the forearm muscles are not wasted), and the rotation of the scapula from the loss of the trapezius and rhomboids. Fig. 170 shows the habitual appearance of the face, and Fig. 171 the greatest possible movement of the facial muscles in closing the eyes and smiling, and also the wasting of the deltoids and pectorals. The general distribution of the atrophy and projection of the scapulæ are seen in Fig. 172.[*]

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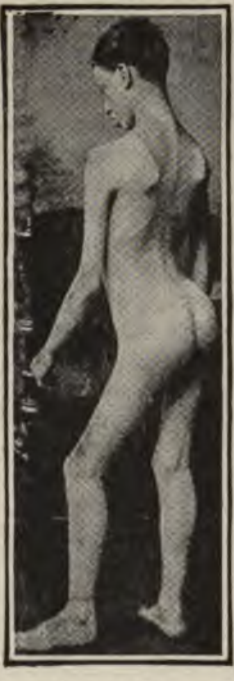
[*] The patient was a lad sixteen years of age. The following is an abstract of the notes of his case. In his family no history of any similar affection can be ascertained. There is conclusive evidence that he had a hard chancre at the age of three, followed by secondary symptoms. The muscular wasting began gradually during childhood; even when very young there was little movement in the face, and that ceased at the age of six or seven. Walking became difficult about three years ago. The face is almost motionless: an effort to smile causes only a just perceptible movement of the left cheek, the amount of which is fairly indicated by the difference between Figs. 170 and 171. It is accompanied by a distinct movement of the ears. The lips are full and can be brought together, but not shortened. The eyelids cannot be made to meet, even with a strong effort, being then $\frac{1}{8}$ inch apart. Forehead absolutely motionless. Eyeballs prominent, movements normal. Masseters, tongue, pharynx, and larynx unaffected. In the neck the sterno-mastoids are very small and feeble, the omo-hyoids large and strong. The following muscles seem quite gone—

FIG. 172.



begins in childhood its progress is sometimes very slow. Thus in one case the affection of the face was first noticed at five, at twelve the arms began to suffer, and a few years later the flexors of the hip became weak, but the patient was still able to walk at the age of forty. The disease may increase until development is over and then remain stationary, as in a case in which, at forty-four, there had been no change since the age of twenty. The patient's daughter was more severely affected.* On the other hand, the progress of the disease may be more rapid and uniform, and the atrophy may reach its widest distribution in eight or ten years. In most cases, even of severe type, the wasting does not become universal, but remains limited to the muscles mentioned above, but occasionally hardly any muscles of the body may escape.

The duration of the disease varies from ten to fifty years. Death has never occurred as the direct result of the malady. In the cases of most severe degree and rapid course the patient has usually died from phthisis, probably related to the deficient breathing power, just as in the sufferers from pseudo-hypertrophic paralysis. In most cases, however, death has been due to other maladies, and has not been in any degree the consequence of the muscular disease.

PATHOLOGICAL ANATOMY.—In general, the condition of the muscular fibres resembles that met with in pseudo-hypertrophic paralysis, the increase of interstitial tissue being wanting except in those

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trapezius (except, perhaps, a little of the middle part of the right), rhomboids, deltoid, pectorales, latissimus dorsi, serrati, biceps, brachialis, triceps, supinator longus. The lev. ang. scapulæ, supra- and infra-spinatus are unaffected; the latter is large, but probably only hypertrophied from over-use. The supinator longus is feeble on the left, powerless on the right side. The extensors of the phalanges of the thumb are lost on the left side, and the extensor carpi radialis on the right. All the other forearm and hand muscles appear to be unaffected. The erectors of the spine are small and feeble; intercostals strong, diaphragm powerless, abdominal muscles weak. In the legs the glutei and flexors of the knees seem normal, the flexors of the hips weak, extensors of the knees small and feeble, muscles of the lower leg rather small and feeble, but only the peronei are powerless, right-angled contracture of ankle-joint with tendency to varus. Knee-jerk absent. Circumference (in inches) of upper arms (middle), R. $5\frac{15}{16}$, L. $5\frac{7}{8}$; of forearm (maximum), each 8; of thighs (minimum $2\frac{1}{2}$ inches above patella), R. $10\frac{1}{4}$, L. $9\frac{1}{2}$; calves (maximum), R. $10\frac{3}{4}$, L. 11. Electric irritability much lowered in all affected muscles to each current; in most, no contraction can be obtained. Sensation, the sphincters, and the visceral functions are unaffected.

* Landouzy and Dejerine, loc. cit.

cases that really belong to this form, but in which the characteristic enlargement is absent, because no fat-cells are developed in the tissue. A multiplication of nuclei is, however, sometimes seen. The chief change is sometimes a simple narrowing of the fibres, with an ultimate disappearance of the transverse striation. Degenerative changes are frequently met with, indistinct striation or granular, fatty, or "waxy" transformation being seen here and there, alike in narrowed or enlarged fibres, and in those that retain their normal calibre; occasionally fibres present longitudinal striation, fissuring, or vacuolation; but in other cases, or in some muscles (especially in the "juvenile" form), a remarkable increase has been found in the size of fibres in excised fragments,* even of muscles that are below the normal size. It seems to show definite pathological enlargement.

The statements made regarding the spinal cord in the pseudo-hypertrophic form are strictly applicable also to this variety. It is normal as a rule, to which exceptions are so rare that they can have no relation to the malady, save that of secondary indirect consequences. The nerves also are normal, if the cases are eliminated in which the symptoms indicate the peroneal form.

Thus the facts ascertained regarding this type do not suggest any special addition to the general conclusions intimated in the introductory paragraphs. The affection appears to be the result of a primary developmental tendency, involving the muscular tissue only, and cannot be regarded as a secondary result of the interstitial changes, except in a trifling, occasional, and unimportant degree. Still less is it a consequence of changes in the spinal cord. If it is in any measure the result of failure in the nutrition of the terminal structures of the nerve-fibres (by which the nervous and muscular tissues are connected, and which is perhaps to be regarded as intermediate between the two), the fact has yet to be demonstrated, and its significance ascertained.

DIAGNOSIS.—The two most important diagnostic indications are the affection of more than one member of the same family, and the onset of the disease before adult life is reached. The former is practically conclusive; and the latter should always suggest the probable idiopathic nature of the case. In isolated cases the diagnostic difficulty is much greater, and no indication is actually conclusive, except, perhaps, the affection of the face. Commencement during childhood is also of great weight. It is most unlikely that progressive muscular atrophy, beginning under ten, is of spinal origin, but I have known such atrophy, certainly spinal, to commence at the age of fourteen.† The peculiar affection of the zygomatic muscles is very characteristic, but we cannot yet say that it is pathognomonic (see note on p. 587), and the affection of the lips must be carefully distinguished from that due to the bulbar palsy so commonly associated with spinal atrophy. Of

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* See especially Schulze, 'Ueber den mit Hypertrophie verbundenen Muskelschwund,' Wiesbaden, 1886, and Hitzig, loc. cit.

† See, however, p. 597.

[illegible] wasting, that of the latissimus and lower half of [illegible] of the hand muscles are the most im- [illegible] although they are not by themselves con- [illegible] sometimes met with in spinal atrophy.

[illegible] important distinctions between spinal and idiopathic [illegible] they are available, depend upon the symptoms of [illegible] or of the bulbar nuclei. Especially significant [illegible] knee-jerk which results from lateral sclerosis, a [illegible] of spinal disease, rendering the spinal origin of [illegible] certain. The distinction from the pseudo-hyper- [illegible] already considered; that from the peroneal type [illegible] mentioned.

[illegible]—The extreme variations in the course of the disease [illegible] in any individual case both uncertain and difficult [illegible] Speaking generally, chronicity favours arrest, and the [illegible] appear the slower will be their progress. The [illegible] less grave in the simple atrophy than in the [illegible] variety of the pseudo-hypertrophic disease, and in [illegible] number of cases, perhaps one half, the malady has not [illegible] life. Even in cases that begin during youth it is [illegible] that the patient may reach old age. It seems also [illegible] the cases that do not involve the facial muscles.

[illegible]—It is not yet clear that idiopathic muscular atrophy [illegible] influenced by treatment in any considerable degree. Most [illegible] of cases are almost silent on the subject, and the [illegible] that few individuals have an opportunity of forming [illegible] It might be assumed that the essential cause of the [illegible] congenital tendency, withdraws it from the range of thera- [illegible] the course of the malady is very different from that of [illegible] which own a similar cause. The extreme variations [illegible] the fact that the disease may not be manifested [illegible] and the long period that may intervene between its [illegible] all suggest that other influences co-operate with [illegible] tendency in determining the development of the malady. [illegible] therefore, that the first inference from the history [illegible] may not be altogether correct, although it must be [illegible] that we have not as yet any evidence that the disease is [illegible] influence from drugs. Electrical treatment and mass- [illegible] been thought to do good and even to produce arrest (Erb), [illegible] tendency of the malady renders the evidence of arrest [illegible] voluntary exercise is practicable, this is a far more [illegible] to muscular growth than any electrical application, [illegible] that such exercise, carefully persevered in, may do [illegible] the occurrence of the malady in those pre- [illegible] even to retard its progress in those who are [illegible] In this connection it is noteworthy that very few [illegible] engaged in occupations that involve active muscular exertion. Over-exertion should, however, be carefully avoided. The general health should be attended to, and any defect removed as speedily as possible, both in those who suffer and in those who are related to sufferers, since, as we have seen, depressing influences may apparently excite the development of the disease, and it is therefore reasonable to suppose that they may accelerate its course, and lessen any tendency there may be for the morbid process to become stationary.

The Peroneal Type of Family Amyotrophy.

(*Neuritic Muscular Atrophy.*)

The name "peroneal type" has become current as the most convenient designation for the variety of muscular atrophy, occurring in early life, to which it was applied by Dr. Howard Tooth in a Thesis published in 1886.* This was the means of calling general attention to the form, of which, however, an account had been published shortly before by Charcot and Marie,† and many cases had been previously described, in most instances without recognition of their special character.‡ The cases of this form present certain resemblances to the idiopathic atrophies just described—resemblances sufficiently important to have led to the description of these cases in association with the primary myopathies, embracing, as they do, the age of the sufferers, the occurrence of the disease in members of the same family, its gradual onset, and its very slow but progressive course. Yet this position must be regarded as provisional only, and probably erroneous. The distribution of the wasting involves a constant difference from the idiopathic muscular affections, and some of the cases, otherwise inseparable from the rest, present features that indicate neuritis. The condition may have, as an antecedent, an acute specific disease—a circumstance that has the same significance. The facts ascertained concerning this form must, therefore, be carefully excluded from our generalisation regarding idiopathic atrophy.

In this disease, males suffer about twice as frequently as females. It generally begins in the second half of childhood, and very seldom after twenty, although cases have been described beginning as late as forty. It is occasionally hereditary, and still more frequently collaterals suffer, several brothers and sisters being affected. The wasting is first apparent in the extensor longus hallucis, or extensor communis digitorum, or in the peronei muscles. According to Tooth it very often begins in the latter, and occasionally in the gastrocnemius, but it is probable that it occurs simultaneously or even earlier in the small muscles of the foot, where it readily escapes observation. Such early atrophy in the feet has, indeed, been actually found in some cases that came under observation at an early stage.[*] The calf muscles suffer subsequently, and still later those of the thigh, especially the vastus internus. The unequal affection of the muscles of the leg causes the early development of club-foot, which, especially in children, becomes a characteristic symptom.[†] The arms are only invaded, in most cases, some years after the legs, so slow is the course of the disease. The intrinsic muscles of the hands (thenar, hypothenar, and interosseal muscles) are first attacked, and subsequently those of the forearm: sometimes the extensors and sometimes the flexors suffer first and most; while the supinator longus remains normal, and so do the muscles of the shoulder, neck, and back. The affection, however, does not necessarily commence in the lower limbs, and the face may be affected (Hoffmann). The atrophy is often symmetrical, but occasionally the muscles on one side waste first and most. The claw-like deformity of the fingers may result from the affection of the intrinsic muscles of the hand. This is so rare in early life from any other cause that its significance is considerable, and almost distinctive when combined with the deformity of the feet just mentioned. The case shown in Fig. 173 was probably an example of this type.[‡]

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* 'The Peroneal Type of Progressive Muscular Atrophy,' London, Lewis, 1886.

† 'Revue de Méd.,' 1886, p. 97.

‡ By Friedreich, Eichhorst, Oppenheimer, Ormerod, and others. A table of thirty collected cases (some doubtful) is given by Tooth.

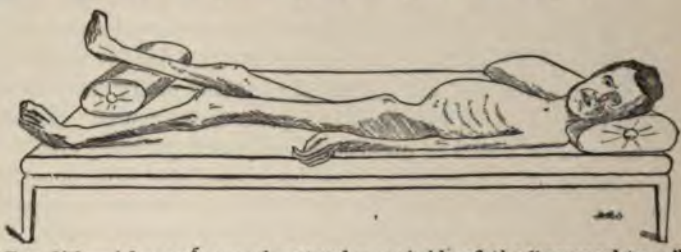

FIG. 173.—Advanced muscular atrophy, probably of the "peroneal type." (Drawn by Dr. Spencer, from a photograph.)

The affected muscles (especially in the hand) occasionally present slight fibrillation. Their electric irritability is diminished sooner, and in greater degree, than in the ordinary idiopathic atrophy, while a still greater difference is presented by the fact that faradic irritability may early become extinct, and that there may be a distinct reaction of degeneration. In connection with this should be noted the important fact that cutaneous sensibility, though often normal, is sometimes impaired or lost, especially over the region in which the atrophy is greatest, or upon the soles of the feet. Pains occur in some cases. and so also do spontaneous spasmodic contractions, especially in the muscles of the thigh. The muscle-reflex action is lessened or lost in the affected regions, but there may be extensive atrophy below the knees, without loss of the knee-jerk, if the thigh muscles are preserved. Cutaneous reflex action presents considerable variations, and is preserved more often than would be expected. Vaso-motor disturbance sometimes occurs (Sachs).

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[*] Hoffmann, 'Arch. f. Psych.,' xx, p. 660; Joffroy, 'Gaz. Hebd.,' 1886, No. 18.

[†] See Sachs, 'Brain,' Winter part, 1889.

[‡] It was given in the first edition as an example of simple atrophy. The patient was a man aged twenty-seven at the time of his death (in University College Hospital). No history of any analogous case in his family could be ascertained. The affection began at the age of fourteen, when his feet began to turn in so that he walked on the outer side of the foot, and soon he noticed gradual wasting of the legs, which slowly progressed, and about the age of twenty-four the arms also began to suffer. When first seen, a few months before his death, the muscular atrophy was universal, and the subcutaneous fat had also disappeared. Even the hands were greatly wasted; there was a hollow in the position of the thenar eminence, just as in progressive muscular atrophy, and the interosseal muscles were also greatly wasted. There was slight fibrillation. The intercostals were paralysed. The wasting of the legs was extreme; the maximum circumference of the calf was

The nature of these cases has still to be demonstrated. Their very slow course, the time of life at which they begin, and the affection of several members of the same family, are features analogous to the idiopathic atrophy; but a marked contrast to this is presented by the tendency of the malady to succeed an acute specific disease, measles, and this also may appear as a family tendency. This feature resembles the causation of multiple neuritis, and the electrical reaction of degeneration is conclusive proof of preponderant changes in the motor nerves, while anæsthesia demonstrates that, in some cases, the sensory fibres are also affected. In such cases a peripheral neuritis, motor or total, of extremely chronic course and peculiar origin, must be recognised as the only possible lesion, and it was actually found, in one case by Friedreich, and in another by Gombault. Dubreiulh[*] also, in a case which he examined, found that there were inflammatory changes in the nerves, the spinal cord being normal. If this is the cause of the symptoms in some instances, the question arises whether it is not the lesion in all the cases of this peroneal type. Such a lesion is conceivable; the degenerative form of neuritis presents every degree of chronicity, and may be limited to the motor fibres and to certain muscles. This pathology was advocated as the probable one by Tooth, who pointed out that it would also enable us to understand the occurrence of fibrillation, which is extremely rare in the idiopathic atrophies. It is, indeed, asserted by Hoffmann that not only are the peripheral nerve-fibres the seat of a primary degeneration, but that this change ascends the nerves to the anterior and posterior roots, and involves, in a secondary manner, the elements of

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only seven inches; the feet were everted, the sole hollowed, and the toes flexed. The patient died from diarrhœa. The muscles were found to be small and pale. The chief microscopical change was intense granular and fatty degeneration of the fibres. A very few normal fibres were seen in some muscles. There was no increase of interstitial tissue. Microscopical examination revealed no morbid changes in the spinal cord. The nerves, unfortunately, were not examined.

[*] 'Rev. de Méd.,' 1890; 'Neur. Cent.,' 1891.

the spinal cord, especially the fibres of the posterior median columns (which continue the path from the muscles) and the motor cells of the anterior cornua. But we do not know how far the premature failure of nutrition is a secondary process, transmitted from below, or how far it is the result of a defect in vital energy, shared by the central as well as the peripheral nerve-elements.

The intimate relation between the motor nerves and the muscular fibres should also be kept in view in this connection. There may be a morbid tendency in the nerves similar to that of the muscles, including a liability to early slow degeneration, analogous to that which the muscles present, and equally prone to occur in several members of a family. But it is desirable that the neuritic nature of the affection should be placed on a broader basis of ascertained fact before it is allowed expression in the name given to the disease.

It should also be remembered as possible that the atrophy may be idiopathic in some cases, while in others it may be secondary to such a neuritis as above mentioned. There is nothing difficult, or even anomalous, in the conception of a congenital tendency to premature failure of nutrition in the peripheral nerve-fibres, like that which, in the posterior column and other fibres, seems to underlie "hereditary ataxy," or which, on the other hand, in the muscles is the apparent cause of idiopathic atrophy. Further, if the lesion is really neuritic, we can understand that an acute specific disease like measles should occasionally be its excitant, although its occurrence in families shows that a congenital tendency must be called into activity by the virus, and the cases must be distinguished from those in which a pure neuritis is induced by the influence of the poison of the specific disease. An acute specific disease, the virus of which is known to be capable of inducing the degeneration of "parenchymatous" neuritis, may readily excite this state in each of several members of the same family if a congenital tendency to it exists in them. It may readily evoke that which it could, even alone, produce. According to this theory, a tendency to nerve degeneration, acting alone, may lead to the early failure of nutrition of the nerves, while the wasting of the muscular fibres may be either a consequence or a concomitant, in part or altogether. Thus the muscles may atrophy because the nerves have degenerated, or because they also have a tendency to atrophy, or from both causes combined. Nerve and muscle may alike share the congenital imperfection in some cases, while in others its primary incidence is on the nerves alone, the muscles suffering in consequence of the changes in the nerves. The congenital association may sometimes be even more extensive, and a tendency to the early failure of structures in the central nervous system may be conjoined, giving rise to cases of complex aspect and character.

It is especially important to note, in this connection, the occurrence of cases that occupy an intermediate position, and combine the characters of this and the myopathic types. Such combined cases are usually partial only, but such partial cases, when combined, ultimately cover the whole intermediate ground. For instance, the condition of the legs in the "peroneal type" has co-existed with that of the arms in the "juvenile form" in several recorded cases.*

Thus the possibility must be recognised that the cases of this type vary, even more than their characters suggest, in the degree in which the morbid tendency acts upon nerve or muscle, or both, as well as the degree in which its activity is spontaneous or induced. Hence the cases may here be only partly out of place—some, perhaps, not at all; and their position, as forms of "neuritis," might be equally open to exception.

The progressive character of the cases of this type is commonly pronounced; and, even if slow, it renders the prognosis gloomy, and the scope for treatment small. So far as anything can be done, it is by the measures already mentioned in the account of the treatment of the maladies to which the cases approximate, the primary diseases of the nerves on the one hand, and of the muscles on the other.

Family Form of Muscular Atrophy in Children with Spinal Lesion.

The above seems to be the most suitable title for a series of cases described in recent years by Werdnig † and Hoffmann ‡ in Germany, and Bruce and John Thomson § in this country. The condition is marked by the occurrence of similar atrophy in more than one member of the family, although in Bruce and Thomson's case the condition was an isolated one. It is characterised by the onset of weakness, especially in the hip and back muscles, about the age of from seven to twelve months, in children previously healthy. The weakness gradually impairs and finally abolishes the power of walking, and similar weakness usually attacks the upper limbs as well as the legs, and even the small muscles of the hand may be affected. The condition is one of marked atrophy without sensory impairment, although in one case there was a curious insensibility to the pains of faradic stimulation of the affected muscles. In many of the muscles the reaction of degeneration may be present, and occasionally bulbar symptoms are superadded. The condition culminates in one of extreme helplessness, with marked wasting of muscles without any fibrillary twitching, although death did not take place in some cases until the age of six was attained. No hypertrophy has been noted in any case.

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* *E.g.* by Hoffmann, 'Berlin. klin. Wochenschr.,' 1887, No. 22; and Eisenlohr, 'Neur. Centralbl.,' 1889, p. 565.

† 'Arch. f. Psych.,' 1891 and 1894.

‡ 'Deutsch. Ztsch. für Nervenheilk.,' 1893 and 1897.

§ 'Edin. Hosp. Reports,' 1893.

The conditions found after death were marked atrophy of muscular fibres, although occasionally some fibres were hypertrophied. Atrophy also of nerves and anterior roots was found, and wasting of the cells in the anterior horns. The latter was distinct, and this, with the absence of any hypertrophy at any time (although one child had been noted as very fat which afterwards became much wasted), marks off the cases distinctly from those of pure muscular dystrophy. The type possibly, like the peroneal, forms a connecting link between cases of spinal progressive muscular atrophy and those of true myopathy without any spinal lesion. The presence of definite changes in the cells of the anterior horn and the anterior nerve-roots connects the cases with the former, while a connection with the latter is formed by the simple wasting with occasional hypertrophy of isolated muscular fibres, and by the occurrence of the disease in more than one member of a family.

DIAGNOSIS.—The age at which the disease occurs, the absence of fibrillary twitching, and the early suppression of the reflexes, as well as the frequent occurrence of more than one case in the same family, will sufficiently distinguish these cases from ordinary progressive muscular atrophy. On the other hand, the absence of any hypertrophy, the affection of the small muscles of the hand, and the occasional presence of bulbar symptoms will mark them off from cases of true muscular dystrophy. In every case which comes under notice hypertrophy in some muscles, especially the infraspinati, should be carefully looked for, as it may turn out that the cases are more closely connected with myopathic cases than is as yet apparent from the evidence.

PROGNOSIS.—This seems to be extremely bad; the cases go on from bad to worse, the weakness becomes greater, the atrophy more pronounced, until death occurs. But, as has been stated above, this has not unfrequently been delayed as late as the sixth year.

TREATMENT.—The nature of such cases, indicating as they do an inherited tendency to early death in the muscular fibres and their correlated nervous structures, precludes the hope of successful treatment. The administration of strychnia hypodermically is indicated, although the age of the patient renders extreme care necessary in the choice of doses. It is also possible that galvanism, combined with massage and passive exercise, might have a beneficial effect. And, of course, care must be taken to ensure wholesome and judicious feeding and satisfactory hygienic conditions.

PERIODIC FAMILY PARALYSIS.

Under this name Goldflam* has described a condition of which examples had been previously published by Westphal, Cousot, and Schachnowitsch. It is characterised by recurrent paralysis commencing usually in the legs, involving also the arms, and passing off in the reverse order after a period varying from twenty-four hours to three days. During the attack of paralysis the patient is sleepy, but there is no impairment of consciousness, sensibility is retained, and the paralysis is flaccid, with diminished knee-jerks and loss of plantar reflexes. Speech and swallowing are not interfered with. In Goldflam's case there was great diminution of faradic irritability in the upper limbs, and complete loss in the lower limbs. After the attack had passed off the nerves in both arms and legs reacted normally. Eleven members of the family suffered from similar attacks of paralysis. They began between fifteen and twenty, and increased in number as age advanced, although the individual attacks did not last so long. In the patient whose case is particularly described there were only two attacks in the first year, but after three years they occurred every fortnight.

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* Berlin Congress, 1890; 'Neur. Cent.,' 1890, p. 638.

The condition as regards etiology is obscure. Goldflam's opinion is that it is the result of auto-intoxication—a view which is favoured by the fact that in his patient obstinate constipation persisted during each attack of paralysis.

Apparently the disease does not in any way tend to shorten life, but treatment is of little if any effect.

MUSCULAR HYPERTROPHY.

The occurrence of a true hypertrophy of muscles, as a condition of disease, is exceedingly rare. In most cases in which the muscles are enlarged, the increase in size is due to a growth of interstitial tissue, fatty or fibrous, such as has been described in the chapter on pseudo-hypertrophic paralysis. We have seen that muscular fibres larger than normal have been described in that disease, and also in some cases of simple atrophy. When this increase in size has been met with only in excised fragments, it is possible that the condition may have been due to a vital contraction excited by the process of excision, and such evidence of hypertrophy is certainly inadequate;* only when it is found after death can its occurrence be regarded as beyond question. Nevertheless true hypertrophy of fibres is a change that may be expected to be occasionally met with in cases that are due to a congenital tendency, and its presence, even in muscles that are the seat of atrophy, need, therefore, excite no surprise. It was met with by Bruck in a remarkable case of general muscular enlargement in an idiotic child, in whom it apparently developed after birth. The affection first showed itself in the tongue, but the muscles presented spasmodic contractions, and the case, like most of the kind, was evidently anomalous in its features. Muscular hypertrophy has been found in the singular malady described in the next section (Thomsen's disease). It has also been met with, in very rare instances, as a wide-spread or partial condition, usually associated, strange to say, with either diminished power or with a morbid readiness of fatigue.* The muscles most frequently affected have been those of the shoulder and upper arm, or of the thigh and calf, on one side or on both. It has also been observed in the glutei, spinal muscles, and trapezii. The causes are obscure, but it has been ascribed to over-exertion. The diameter of the fibres has been increased to double the normal, a maximum of $\frac{1}{200}$ inch having been met with (Eulenberg), whereas the normal maximum may be taken as $\frac{1}{400}$ inch. An increase of the nuclei has been observed, without any overgrowth of the interstitial tissue.

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* Since the above was written, the doubt there expressed has been confirmed by an observation made for me by Dr. H. R. Spencer. A fragment of the gastrocnemius of an amputated leg was excised immediately after the amputation, and the fibres were compared with those of the muscle twenty-one hours later. In order to ascertain more definitely whether a vital contraction persisted as an apparent increase in size, a part of the excised fragment was separated and faradised. The average size of the fibres in the muscle was $\frac{1}{740}$ inch, in the fragment excised $\frac{1}{625}$ inch, in the fragment faradised $\frac{1}{417}$ inch. Since the division of the nerve during the amputation may have caused some contraction, it is possible that the difference in size produced by excision may be even greater than these figures represent. [I leave the above note unchanged because, since it appeared in the first edition, the observation and doubt have been fully confirmed by various observers, *e.g.* by Oppenheim and Siemerling, 'Med. Centralbl.,' 1889, No. 39.] It is interesting, however, to note that enlarged muscular fibres have been found in the atrophied muscles in infantile paralysis.

The condition is manifested by an increase in the size of the muscles, which are also firm. The circumference of the limb is greater than normal, and when the change is unilateral the difference between the limbs of the two sides may be very striking. The maximum circumference of the calf has been as much as seventeen inches. The muscles are soon exhausted, and have been weak in some cases, while in others there has been abnormal strength for brief exertion. The electrical, mechanical, and myotatic irritability of the muscles has usually been found unaltered.

The disease may be suspected if a marked increase in size in an adult is accompanied by impaired power of sustained exertion, if the muscles are firm, and the patient does not manifest other indications of pseudo-hypertrophic paralysis. The diagnosis can, however, only be made with certainty by the microscopical examination of an excised fragment, and even then subject to the reservation mentioned on the previous page. The meagre facts regarding the course of the disease suggest that it usually persists without getting either better or worse. Treatment appears to have but little influence upon it.

A singular case has been reported by Eulenberg,† which differs in many particulars from the form of disease above described, and illustrates the complex relations of enlargement of the muscular fibres and its connection with degenerative processes. A man aged thirty-six presented an enormous enlargement of the muscles of the left leg, which were soft and flabby and weak, with lowered irritability. The condition had slowly developed after a fall on the back at ten, which caused imperfect paraplegia, motor and sensory. A year later, during pleurisy, he had thrombosis of the left femoral vein. In an excised fragment of the gastrocnemius the muscular fibres were large, the maximum being $\frac{1}{276}$ of an inch; they presented fatty and vitreous degeneration, and fat was seen between the fasciculi. The muscles of the other leg were somewhat wasted, but the fibres were also enlarged and degenerated. It would appear as though the condition had resulted from a traumatic lesion of the spinal cord, and had been intensified in the left leg by the influence of the thrombosis and resulting vascular disturbance.

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* Auerbach, 'Virchow's Archiv,' Bd. liii, pp. 234 and 397; Berger, 'Deut. Archiv f. klin. Med.,' Bd. ix, 1872, p. 363; Friedreich, 'Ueber Prog. Muskelatrophie, &c.,' 1873; Eulenberg, 'Real-Encyclopädie,' Bd. ix, p. 354. A very similar case is described by Pal, 'Wien. klin. Wochenschr.,' 1889, No. 10.

† 'Deutsch. med. Wochenschrift,' 1885, No. 12.

THOMSEN'S DISEASE; MYOTONIA CONGENITA.

The malady thus designated may be considered here, notwithstanding the obscurity of its nature, because it agrees with the diseases last described, in that the symptoms are muscular, and that the disease seems often congenital and occurs in families. It has been named after the physician whose description gained for it general notice, and who is himself its subject, but it had been previously described by Leyden, and hinted at, long ago, by Sir Charles Bell.[*] It is not always congenital, and "Transient Myotone" would be a more exact name.

The disease is characterised by a peculiar rigidity of the muscles, which comes on when they are first put in action after a period of rest. The rigidity is transient, and when it has passed off, it does not return

[*] Bell's 'Nervous System,' Case 184, p. 436. The objections to cognominal nomenclature of disease are certainly reduced to a minimum when the first describer of a rare disease is also its subject. The most important papers on the disease are those of Leyden, 'Klinik der Ruckenmarkskr.,' 1874, Bd. i, p. 128; Thomsen, 'Arch. f. Psychiatrie,' Bd. vi, 1876, p. 702, also 'Centralbl. f. Nervenkr.,' 1885, p. 193; Bernhardt, 'Virchow's Archiv,' Bd. lxxv, 1879, p. 516, and 'Centralblatt f. Nervenkr.,' 1885, p. 122; Ballet and Marie, 'Arch. de Neur.,' 1883, No. 13; Möbius, 'Schmidt's Jahrb.,' Bd. cxcviii, 1883; Ringer and Sainsbury, 'Lancet,' 1884, pp. 767, 816, and 860; Erb, 'Die Thomsen'sche Krankheit,' Leipzig, 1886, also 'Deut. Archiv f. klin. Med.,' 1890, xlv, p. 529; Seifert, ib., xlvii; Marie, in 'Encycl. des Sc. Méd.,' 1886; Hale White, 'Brain,' April, 1886, and 'Guy's Hosp. Rep.,' vol. xlvi (a very valuable paper, with bibliography); Jacoby, Journ. of Neur. and Ment. Dis.,' 1887, xiv, p. 23; Jolly, 'Südwest. Neurol.,' Baden, June, 1890; Buzzard, 'Lancet,' 1887.

as long as exertion is continued. The malady is often hereditary, and usually affects several members of the same family. In that of Thomsen, cases can be traced through five generations. It appears to be more common in Scandinavia and Germany than in France or England, but it is a rare disease;* and although a considerable number of clinical cases have now been recorded, there has been no post-mortem examination. There is little to attract attention in the slighter forms, and the rarity of these may ultimately be found to be less than at present appears.

Both sexes suffer. In most cases the symptoms have been first noted in childhood, between four and ten years of age, sometimes even in the cradle. They increase during the period of muscular development, and then remain stationary. A few patients have seemed free until about the period of puberty, but even in such instances it is probable that the tendency was congenital in origin, since the cases occurred in families, other members of which suffered earlier. But in a few cases symptoms apparently identical with those of the congenital disease have come on in early manhood after some exciting cause—in one case a lightning stroke, in another a sudden alarm.† Severe and prolonged exertion during two years preceded the onset of very characteristic symptoms in a man of twenty-five, without heredity.‡ In such cases the disease has been apparently acquired.

The characteristic symptom is tonic spasm of the muscles when they are put in action after a period of rest. As soon as the patient attempts to move, the muscles become rigid. The rigidity may make movement impossible while it lasts, or may merely lessen the possible range of movement. After a few minutes, or less, the spasm passes away, to be renewed, but in slighter degree, by a fresh attempt. If the movements are continued, the spasm soon becomes trifling, and does not return until after a period of rest. The longer the rest, the more troublesome is the spasm. The subject can walk for hours without fatigue when it has passed off. The rigidity is sometimes lessened by alcohol and increased by attention and by fear of it; the more the sufferer tries to overcome the stiffness, the less is he able to do so. It is also worse in cold and damp weather. In a severe case, a slight impulse will make the person fall, and it may then be impossible for him for some minutes to rise from the ground. The arms are usually less affected than the legs, but in some cases the rigidity fixes the fingers for a short time on an attempt to use the hand. The muscles of the face are usually free; mastication may, however, be interfered with by the spasm in the muscles of the jaw. Rarely, the tongue or face has been affected; still more rarely the muscles of the eyeball, interfering with its movement and retarding the descent of the upper lid.[*] Most voluntary muscles, indeed, seem liable to suffer, even the muscles of the larynx and pharynx; those concerned in the processes of respiration, defæcation, micturition, and coitus have also been involved. Sometimes the spasm is greater on one side of the body than on the other.

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* In England cases have been observed by Buzzard, Herschell, Hale White, Benham, Chapman, and others.

† Engel, 'Phil. Med. Times,' 1883, p. 412; Schönfeld, 'Berlin. med. Wochenschr.,' 1883, No. 27.

‡ Moyer, 'Med. News,' 1890.

The muscles are always well nourished; they are often, indeed, above the normal size, and possess more than normal strength, but they are sometimes less strong than is normal, even when large. Thomsen believes that the more the muscles are employed the less severe is the spasm, and that a life of active exertion produces some permanent amelioration in the disease. The malady is, however, a source of some disability, and of considerable annoyance; in the words of Thomsen, "it casts a shadow over the lives of the sufferers," and may have caused the mental irritability and hypochondriacal tendency that have been conspicuous in some cases.

Careful investigations of the muscular phenomena have been made by several observers, and that by Erb is especially instructive. In his case a single brief effort caused tonic contraction which lasted for twenty-five seconds. Momentary electrical stimulation of the nerves caused only a momentary contraction of the muscles, but continued stimulation always causes a prolonged contraction, the "myotonic reaction" of Erb. In certain muscles, moreover, an uninterrupted current caused peculiar wave-like contractions, about one per second, passing from the negative to the positive pole.[†] Any strong stimulation of the muscle itself caused a prolonged after-contraction.[‡] The irritability of the nerves is generally normal in degree; that of the muscles is normal or increased to voltaism. In some cases the anodal closure contraction has occurred with undue readiness, and the "latent interval" after stimulation has been found unchanged (Hale White) or increased.[§] The muscles are remarkably sensitive to mechanical stimulation, and firm pressure may cause a tonic contraction lasting from twelve to twenty seconds.

In all undoubted cases of this disease sensibility has been intact, and the superficial reflexes have been unaltered. Myotatic irritability is normal or increased.

Once developed, the condition seems to persist, with little change, through life, but in one case, a female (in whose family were other cases), considerable improvement is said to have followed marriage.[||]

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[*] Raymond, 'Gaz. Méd. de Paris,' June, 1891.

[†] Although Erb, Seifert, and others have noted this, some observers (as Hale White) have failed to obtain the phenomenon.

[‡] Jolly has found that repeated stimulations by either current, without too long an interval between the successive stimulations, tend to render the after-contraction less and less evident, until it ceases to occur ('Neur. Centralbl.,' 1890, p. 438).

[§] ·025 or ·03 sec. instead of ·01 (Bluminau, 'Neur. Cent.,' 1888, p. 679).

[||] Herschell, 'Lancet,' Feb. 1, 1890.

In some instances the symptoms are slight, and remain so, even when they begin in early life and occur in the same families as severe cases. Such cases, when isolated, may readily be overlooked.

PATHOLOGY.—An examination of the central nervous system has yet to be made, but it is unlikely to yield results of novelty or importance. The state of the muscular fibres has been ascertained from excised fragments—a method which, however, involves the fallacy, already mentioned (p. 599, note), of possibly stimulating the fibres to contraction. At the same time the evidence of hypertrophy appears conclusive. Where the minimum width was the same as in health, the maximum diameter was double the normal (Erb). In other cases the maximum or even average diameter in fragments excised under chloroform was double that observed after death in unaffected persons; and that the size was pathological was rendered probable by the aspect of the fibres, which presented indistinct striation, fewer "sarcous elements," irregular, non-parallel edges, and sometimes vacuolation. An increase of the nuclei and of the interstitial connective tissue has been found.*

The manifestation of the disease is in a disorder of the functions of the muscles, and most writers on the subject have followed Leyden in regarding the malady as essentially muscular in nature, as consisting in an altered functional condition of the muscular tissue. This opinion is strongly supported by the frequent alteration in electrical excitability (which can hardly be otherwise interpreted than as an indication of a change in the mode of action of the contractile muscular protoplasm), by the structural changes, and also by the interesting experiments of Ringer and Sainsbury, who found that certain salts, such as sodium phosphate, are capable of causing in the frog tonic spasm bearing considerable resemblance to that of Thomsen's disease, and that such spasm persists not only after the nerve has been divided, but after the intra-muscular nerve-endings have been paralysed by curara. This does not prove that Thomsen's disease is muscular in origin; it is a proof that a similar state may be due to the muscular tissue, but no proof that this is the sole element in Thomsen's disease. We must remember that the malady has apparently resulted, in rare cases, from influences acting on the nervous system in adult life, and also that, in the congenital cases, the spasm only develops, as a rule, after some years of normal action. It may thus be an acquired disease, and the case related at the end of this chapter shows that similar spasm may be due to a primary affection of the cord. The spasm is a transient consequence of rest. But "rest" is not inactivity, either in the nerve-cells or muscles. Muscular "tone" and adaptation to posture mean an unceasing flow of nerve-force from the cells, an overflow perhaps, due to the perpetual elaboration of energy which keeps the cells ready for instant response to the voluntary stimulus. The tonic activity then ceases, or at least does not interfere with the different action excited by the will. But the phenomena of Thomsen's disease suggest that the cells respond abnormally to the voluntary stimulus, that this causes at first an increased tonic activity, slowly ceasing as their energy is lessened by action. Wide as is the difference between the muscular and nervous tissues, we must remember that they have some conditions in common. The dependence of the nutrition of the muscles on that of the motor nerve-fibres and cells is a very remarkable fact, and so also is the influence of the functional activity of the cells and fibres in causing a similar condition in the muscular tissue. To say that the two structures are connected is hardly an explanation of the fact. Whatever is the nature of the relation between them, it is at least conceivable that an abnormal functional state, congenital in origin, may be common to the two, and that the peculiar over-action in the muscles may be accompanied by a similar over-action in the ganglion-cells of the spinal cord, and even in the pyramidal cells of the cerebral cortex. It is even conceivable that the condition of the nerve-cells may be the primary change, and that of the muscles may be secondary, although when produced it is in some degree independent, and may be excited independently by local stimulation. Such a theory enables us to understand the two facts above mentioned—the influence of emotion and the acquisition of the disease, each of which seems to be inconsistent with a purely muscular pathology.

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* Erb, Seifert, and also Nearonow, 'St. Petersburg psych. Gesellsch.,' 1889; see 'Neur. Cent.,' 1889, p. 239. Hale White found no increase in the nuclei.

TREATMENT.—No treatment, properly so called, appears to exert any influence on the disease. The congenital malady persists through life; in the cases in which a similar condition has apparently been acquired, it has also been persistent. The only influence that has appeared to Thomsen to ameliorate the condition is a life of active muscular exertion. But it is possible that the influence of therapeutics is not yet exhausted.

Congenital Paramyotone.—Under this name Eulenberg has described * a strange family affection, allied to Thomsen's disease in its general character, although differing very much in its special features, and equally obscure in nature. The malady was widely spread in the affected family, and could be traced through six generations, but appears now to be dying out. Its congenital character was shown not only by its multiplicity, but also by the fact that in some individuals it was manifested immediately after birth. The symptom was tonic spasm, lasting from a quarter of an hour to several hours, excited chiefly by cold, although often by merely slight cold. The rigidity was followed for a time by weakness. The facial muscles were very prone to become thus rigid, especially the orbiculares palpebrarum and oris; and while the contraction lasted the patient was often unable to speak or to open the eyes. The rigidity was slighter in the legs than in the arms, but the subsequent weakness was equally marked. Warmth removed the spasm. There was no persistent loss of power, and no increase of mechanical irritability. The electrical excitability of the nerves was normal; that of the muscles was lowered to each current, and there was an abnormal tendency to tetanic contraction during the passage of the current. Eulenberg speculates that the symptoms may be due to reflex vaso-motor spasm in the muscles, because the diminution of the blood-supply to muscles renders them weak; but it is clearly equally possible that the two certain phenomena, the sensory impression and the muscular contraction, may be directly connected.

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* 'Neurologisches Centralblatt,' 1886, p. 265.

Ataxic paramyotone seems the best provisional designation for an acquired condition of which one case has come under my notice. Persistent tonic spasm, like the transient spasm of Thomsen's disease, was associated with distinct ataxy, and also with weakness and some anæsthesia. The symptoms commenced gradually in the legs, in a healthy man aged 40, without neurotic heredity; it invaded the arms six months later, and increased more rapidly in the arms than in the legs. At 41½ his state was this: a well-built man, with well-developed muscles, unduly firm in all parts except the neck and head. The firmness was due to tonic spasm, which never ceased. It was present on waking in the morning, and through the day interfered with all movements, making them slow and stiff. He rose from a seat slowly and with difficulty; when standing there was slight unsteadiness, increased by closing the eyes, and a slight impulse or stumble in walking would cause a fall, chiefly because the spasm prevented the needed quickness of movement to save him. Prolonged exertion had no influence on the tonic spasm, which opposed passive as much as active movement. Power in arms and legs was lessened, but not considerably. The grasp = 50 and 55 ko. The extensors of the fingers were weaker than the flexors, but could act perfectly although involuntary flexion followed the slow extension. The electrical irritability of the muscles seemed to be normal. Inco-ordination was especially marked in the hands; with eyes shut, buttoning his coat was almost impossible; the attempt was made with thumb and forefinger, the others being flexed, and he could touch his nose only after many wandering failures. On the palms, from the wrist to the tips of the fingers, sensibility was lost to touch, slightly delayed to pain, little changed to temperature. Slight tactile loss extended to the back of the last phalanges; on the back of the hands it was normal. A similar but slighter loss existed on the soles: he often felt as if walking on a rounded surface. The size and weight of objects placed in the extended hand could not be recognised. No trace of the knee-jerk or other evidence of myotatic irritability could be elicited, but the spasm was enough to explain the failure, and its real condition is uncertain. Mind, special senses, and cranial nerves were unaffected.

Unfortunately the course of the affection could not be observed. Its symptoms must have been due to disease of the spinal cord, and they are instructive as showing that muscular spasm, like that of Thomsen's disease, may be thus produced. The time may not have been sufficient for the production of changes in the irritability of the muscles, but we must also remember that differences in the influence on the muscles may be connected with the defective power.

TUMOURS OF THE SPINAL CORD.

Morbid growths within the spinal canal may spring from the membranes, or may grow in the substance of the cord itself. The difference in seat entails some difference in symptoms, but it is nevertheless convenient to consider the two classes together, because they have many symptoms in common, and it is often impossible to carry the diagnosis farther than the existence of an intra-spinal tumour.

Etiology.—The general causes of these growths correspond, for the most part, to those concerned in their production in other situations, and present few peculiarities that merit special mention. Fatty growth outside the dura mater occurs early in life; malignant tumours develop late. Of tumours within the dural sheath, myxomata are chiefly met with in middle life, tubercular growths occur occasionally in childhood, but generally between fifteen and thirty-five (Herter [*]); lipomata are congenital. Males are a little more prone to suffer than females.[†]

Of the diathetic conditions which give rise to tumours elsewhere, only two, syphilis and tubercle, are effective in causing growths which commence within the spinal canal. Parasitic tumours occur, due to the same influences which produce them in other situations. A few rare growths appear to be congenital in origin, due to the abnormal development of germinal tissue, while the more common gliomata within the cord arise from embryonal tissue, which has suffered an arrest of its normal development. Of the causes of other forms of tumour we know practically nothing. Injuries, such as a blow on the spine, have been supposed to be occasional causes, and their influence in rare instances has seemed possible, but the evidence is not so strong as it is in the case of tumours in some other situations. In many cases the first symptoms have immediately followed some exposure to cold and wet, and it seems probable that their influence may have excited secondary processes in the nerve-elements which were already deranged by the growth, and may thus have frequently evoked the first symptoms of the tumour, but it can have had no share in the production of the growth itself. Traumatic hæmorrhages may, however, result in cystic formations, which have been mistaken for new growths.

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[*] Herter, 'Journ. of Mental Dis.,' 1890, has analysed twenty-six cases.

[†] A collection of facts relating to these points, by Mr. Victor Horsley, will be found in the paper written by him and myself on the case of successful removal of a tumour from the spinal cord, 'Med.-Chir. Trans.,' 1889; but the numerical basis is far too meagre to supply more than statistical suggestions. It will be necessary to wait for a considerable time before adequate data are obtained. A very important paper by Allen Starr ('Am. Journ. of Med. Sci.,' 1895, June), deals with an additional series of cases.

Pathological Anatomy.—The growths within the spinal canal may develop outside the dura mater, inside the dura mater, or within the substance of the cord. The extra-dural tumours may spring from the membrane or from the tissue between the membrane and the bone, or may grow into the canal from the outside, through the intervertebral foramina. Subdural tumours may proceed from the inner surface of this membrane, from the arachnoid, or from the pia mater (Fig. 176). The growths in the cord may spring from the pia mater, or may develop in the substance of the cord. They sometimes proceed from the peculiar tissue which surrounds the central canal.

The forms of extra-dural tumours are lipoma, from an overgrowth of the fat which normally exists between the membranes and the bone; and parasitic tumours, chiefly echinococci; but all are rare.[*] Growths also occur that spring from the bones or intervertebral tissue—enchondroma, sarcoma, and cancerous tumours, which have been already considered. Far more frequent are collections of inflammatory products from bone disease, but these do not come into the category of morbid growths.

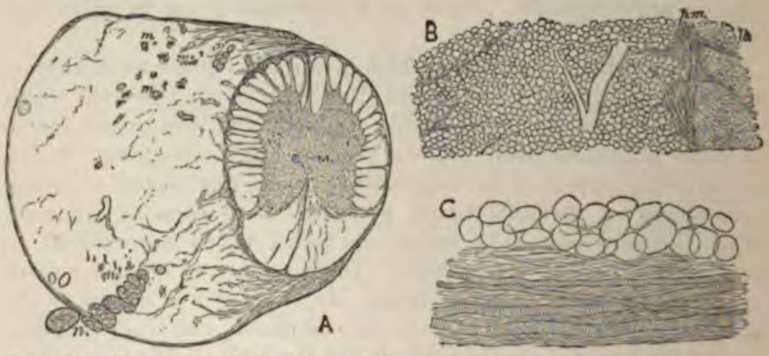

Fig. 174.—Myolipoma of the spinal cord. A, transverse section of the tumour and of the conus medullaris of the spinal cord, to which the growth was attached, and of which the grey matter is represented by the dotted shading c.m. *n*, sections of nerve-roots, partly enclosed in the tumour; *m*, bundles of muscular fibres. B, part of the tumour more highly magnified, showing the fat-cells, of which it was chiefly composed; *p.m.*, pia mater of the cord, of which *l.c.* is part of the lateral column. C, part of the tumour, still more magnified, showing striated muscular fibres, fibrous tissue, and fat-cells (see also Fig. 167, p. 580). The tumour had caused no symptoms.

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[*] An adeno-sarcoma has also been met with (Hodenpyl, 'Am. Journ. Med. Sc.,' 1888).

The tumours which begin within the dura-matral sheath are chiefly syphilomata, sarcomata, and myxomata, sometimes containing cysts or "brain-sand." Tubercular and parasitic tumours are rare, but both echinococci and cysticerci have been met with, developing in the meshes of the arachnoid. Fatty tumours have been found in a few instances, and may contain muscular fibres. They are congenital and sometimes associated with spina bifida (*q. v.*). Fig. 174 represents such a tumour, a myolipoma consisting of fatty tissue and striated muscular fibres, which had grown from the pia mater or meshes of the arachnoid, and was found in a case of tabes.[*] Neuromata occur on the nerve-roots, and may compress the cord.[†]

The growths within the substance of the cord itself are more diverse in character. Syphiloma and glioma are the most common; sarcomata, myxomata, and tubercular tumours also occur, containing the characteristic bacilli, and always associated with tubercle elsewhere. Hydatids are occasionally met with.[‡] Some growths have a compound character, and have been termed myxo-sarcoma, glio-sarcoma, and fibro-sarcoma. Sarcomata and gliomata are sometimes very vascular, and have been termed "angio-sarcoma" and "angio-glioma." It is far more common for the tumour to spring from the pia mater or from the peri-ependymal tissue around the canal than for it to begin among the nerve-structures, but tubercular growths, usually begin in the substance of the cord and usually on one side.

Extra-dural growths are always single; those within the dura mater are often single, but sometimes two or three co-exist; or the growth may be diffuse, as in a sarcoma of the pia mater, which extended as high as the sixth pair of cerebral nerves, and as low as the cauda equina.[§] Neuromata or sarcomata on the nerve-roots are often multiple, and sometimes are very numerous. Neuro-fibromata on almost all the nerves of the body have been observed to coincide with a similar extra-dural growth compressing the spinal cord. Tumours within the spinal cord are also usually single, even those of tubercular nature, but occasionally more than one growth is found.

The size attained by growths outside the cord is necessarily moderate, in consequence of the limitation of the space in which they grow. They vary from the size of a pea to a width of one and a length of two inches, which is rarely exceeded, and chiefly by extra-dural tumours. Those that develop from the pia mater are usually smaller

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[*] See 'Path. Soc. Trans.,' vol. xxvii, 19. Another rare case is one of lymph-angeioma of the pia mater (Traube, 'Neur. Cent.,' 1887).

[†] Myxo-fibromata may also form on the nerve-roots, and even extend on to the cord. See Bruce and Mott, 'Brain,' July, 1887.

[‡] Ransom and Anderson, 'Brit. Med. Journ.,' 1892 (found only after death though operated on), and a case under my care in which the hydatids were found and removed by Mr. Horsley during life. In other cases they have been met with post mortem.

[§] Pasteur, 'Brit. Med. Journ.,' July, 1887.

[illegible] that spring from the dura mater. Multiple tumours of [illegible] nerve-roots are generally small. Adhesions often form between [illegible] and the membrane from which it does not arise. The [illegible] compresses the nerve-roots and the spinal cord (Figs. 175, [illegible]. The compression produced by tumours within the dura mater [illegible] greater than that by tumours outside this membrane. [illegible] of pressure is proportioned to the size of the tumour and [illegible] A soft growth outside the sheath may attain a con- [illegible], and even extend through the intervertebral foramina, [illegible] causing much pressure on the cord itself. Even such exten- [illegible] outside seldom causes a swelling that can be felt. Probably [illegible] rays would yield useful information. At the compressed [illegible] is narrowed and softened, usually indented or flattened, [illegible] pressure is either from one side, from the front, or from [illegible] Rarely the degree of pressure is so great that the cord is [illegible] size of a crow-quill, and it has even been apparently [illegible] at the spot, the upper and lower portions being conical, [illegible] rounded extremities connected only by membranous tissue. [illegible] of the cord depends on inflammation, the "pressure- [illegible] considered in a preceding chapter (p. 390), and it presents [illegible] changes that have been there described. Secondary inflam- [illegible] extend down the cord below the growth, or for a short [illegible] it, often unequally in the different elements—sometimes [illegible] matter into the lumbar enlargement, and even on one side [illegible], see p. 617, *footnote*). The usual secondary degenera- [illegible] conspicuous.

[illegible] that involve the cauda equina often attain a larger size [illegible] occur higher up, because this part of the vertebral [illegible] and the nerve-roots occupy but a small part of it. Most [illegible] this situation are sarcomata or fibro-sarcomata, but it is [illegible] seat of syphilitic growths. They usually spring from [illegible] the arachnoid, and often surround and include the nerves [illegible] the amount of damage to which is very variable.

[illegible] tumours outside the cord are sometimes very numerous. [illegible] usually sarcomata, and spring from the membranes and [illegible] the nerve-roots. They are various in size, ranging from [illegible]-nut to that of a pin's head, and many very small growths [illegible] scattered among the nerves of the cauda equina. In some [illegible] character similar growths have existed in the cerebral [illegible]

[illegible] tumours within the spinal cord are usually small, seldom ex- [illegible] half an inch in diameter, and usually less, even when they [illegible] no considerable impairment of function. But their vertical [illegible] exceeds their transverse diameter, and sometimes they [illegible] through a considerable extent of the spinal cord. Gliomata, and [illegible] the gliomatous growths that spring from the central region, [illegible] from embryonal neuroglial tissue (persistent by arrest of

FIG. 175.

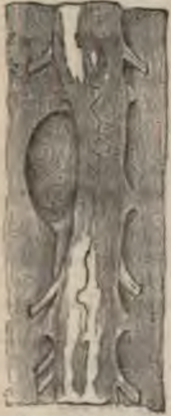

FIG. 176.

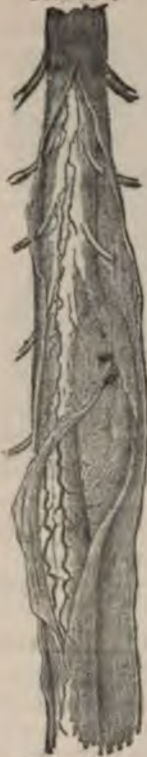

FIG. 177.

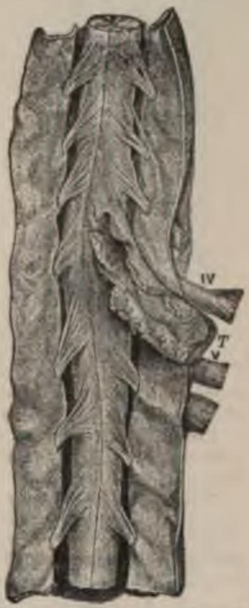

FIG. 175.—Tumour growing from the inner surface of the dura mater, and compressing the spinal cord in the mid-dorsal region. The tumour, 3 cm. long, was a sarcoma in which the cells were arranged in concentric groups, the centres of which had undergone calcification. The spinal cord was softened and dark in tint at the compressed part.* (After Lancereaux.)

FIG. 176.—Sarcoma growing between the arachnoid and pia mater in the mid-dorsal region of the cord. The tumour had sprung from the meshes of the arachnoid, and was only slightly adherent to the pia mater. The spinal cord was compressed and softened. In the figure the dura-matral sheath has been laid open, except at the top, and the arachnoid has been opened over the lower half of the tumour, which, 5 cm. long, lies on the posterior and (right) lateral aspect of the cord.† (After Lancereaux.)

FIG. 177.—A tumour (T) growing from the dura mater, and compressing the right side of the spinal cord at the origin of the 4th, 5th, and 6th cervical nerves. The tumour was a spindle-celled sarcoma springing from the arachnoid.‡ (After Leyden.)

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* The patient was a woman seventy-one years of age, paraplegic, with strong flexor contracture of the legs, loss of power over the sphincters, and considerable impairment, but not absolute loss, of sensibility. The symptoms came on gradually six years before death. (Lancereaux, 'Atlas d'Anat. Path.,' p. 444.)

† The patient was a woman twenty-eight years of age. The first symptom was pain, radiating over the upper part of the abdomen on the right side, and afterwards extending to the leg and to the left side. After a few months, weakness of the legs gradually came on, and became absolute, with loss of sensation and of power over the sphincters. Extensive bedsores were the immediate cause of death, which occurred eighteen months after the onset. (Lancereaux, 'Atlas,' p. 447.)

‡ The patient was a man aged thirty-five. At twenty-eight pain commenced in

FIG. 178.

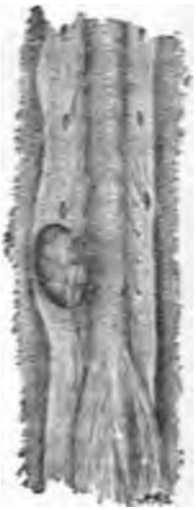

FIG. 179.

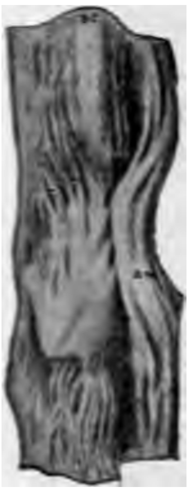

FIG. 180.

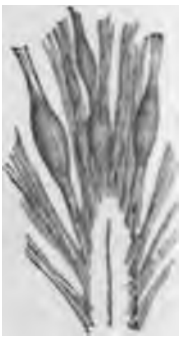

FIG. 178.—Tumour of dura mater opposite the upper part of the lumbar enlargement, compressing nerve-roots and spinal cord. (From a preparation in University College Museum. Drawn by Dr. H. R. Spencer.)

FIG. 179.—Tumour of the cauda equina surrounding and enclosing many of the nerve-roots. The cord itself was unaffected. The structure of the tumour was that of a fibro-sarcoma.[*]

FIG. 180.—Neuromata of the cauda equina. (After Lancereaux.) The growths had apparently given rise to no symptoms.

the right forearm, and continued, intermitting, for some years, until at thirty-four it extended through the whole arm to the neck beside the spine, where it was increased by movement. Similar pain afterwards came on in the left arm. Then followed weakness in the right arm, slight spasm in the right leg, and tingling in the left. The arm became almost powerless and wasted, the right leg weak, and flexion of the neck caused severe local pain. These symptoms continued and increased, and pain in the left leg came on. The cervical spine became tender, and movement of the head to the right was limited. Then both legs became weak, and sensibility was lost as high as the nipples. The paraplegia became absolute, bed-sores formed, and the patient died seven years after the onset of the symptoms. Softening of the spinal cord extended down into the dorsal region. (Leyden, 'Klin. der Rückenmarkskr.,' Bd. i, p. 450.)

[*] The patient was a man aged twenty-eight, whose symptoms commenced nine months before death, and consisted in severe pains in the legs and progressive weakness, the power of standing being lost about four months after the onset. A little power in the flexors of the hips and the extensors of the knees persisted almost to the last, but the knee-jerk was lost. All the muscles of the legs wasted, those below the knee extremely, and even when the patient was first seen electrical irritability was greatly lowered to both currents, without any reaction of degeneration. Tactile sensibility was impaired in each foot and lower leg, more in the right than in the left, and chiefly in the region supplied from the sacral plexus. Sensibility to pain was not affected. The bladder was affected early, and symptoms of pyelo-nephritis already existed. The kidney disease was the immediate cause of death, which was eceded by several convulsions, apparently uræmic. A microscopical examination red considerable damage to many nerve-roots involved in the tumour, while s had escaped. The muscles presented intense granular degeneration with longitudinal striation, and increase of the interstitial nuclei.

development, and often associated with syringomyelia), are those which have most frequently a considerable vertical extent. Such a glioma has been known to reach from the medulla oblongata to the lumbar region, and often extends up from the cervical region to the base of the brain.

The cord is enlarged at the seat of a tumour, and the precise character of the enlargement depends on the position of the growth. In an infiltrating glioma of the upper part of the cord the increase in size has been so great that the wall of the foramen magnum caused a constriction around the swollen cord. Often the abnormal colour of the tumour is perceptible on the surface. The consistence of the cord may be lessened in soft growths, but the tumour is commonly firmer than the normal cord, and the difference is frequently increased by softening in the vicinity of the growth. If the tumour reaches the surface the pia mater and arachnoid may be thickened at the spot, and they may even be adherent to the dura mater, but there is rarely any extension of the meningitis to other parts. On section the growth is usually very distinct, since its aspect contrasts with that of the nerve-substance. Only glioma and myxoma resemble the normal grey substance in appearance, but the new tissue is usually distinct in position. These tumours in some cases blend with the substance of the cord, but in other instances they are bounded by an area of softening which often isolates even invading growths. Some tumours are sharply limited. It may be difficult to say in what structure the tumour began, but this may sometimes be ascertained by an examination of the upper and lower parts, where the growth is commonly limited to that part of the cord in which it first originated. Thus in Fig. 181, B, the tumour occupies a very large area, although

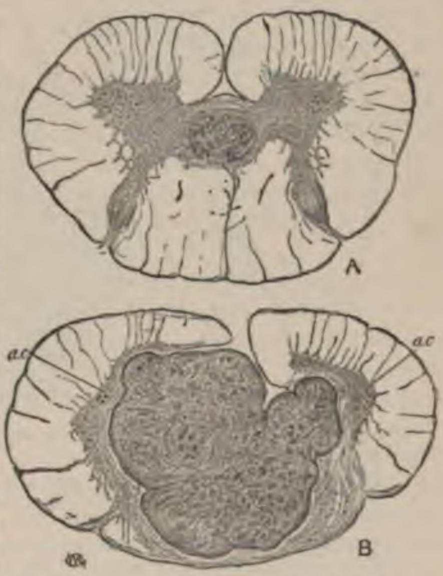

FIG. 181.—Tumour of spinal cord, springing from the tissue around the central canal. A, upper part of the tumour in the middle of the cervical region; the growth is here within the posterior commissure. B, section through the largest part of the tumour, which occupies the whole central region of the cord. The vertical extent was 2 cm. It was a sarcoma composed chiefly of spindle-cells arranged in fasciculi and in concentric layers. The patient was suffering also from a cerebral tumour causing hemiplegia, and the spinal growth, which apparently developed rapidly during the final period of palsy and prostration, was not suspected during life.

the nearly equal extent on each side suggests its central origin, and this is clearly seen in the section through the upper part of the growth, in which the posterior commissure can be traced in front and behind the small area of growth, pointing to the peri-ependymal tissue as its source. Syphilitic tumours commonly originate from the pia mater, as they do also in the brain, and the membrane in their vicinity is often thickened. They both invade and compress the nerve-tissue. An instance of such a gumma of the cord is shown in Fig. 182. It probably commenced at the furrow opposite the head of the posterior cornu, which it has destroyed, and the pressure which it exerted is evident from the displacement of the posterior median septum. In all forms of tumour the central canal is often obliterated by the pressure, and may be slightly dilated higher up; this dilatation is especially common from growths that begin in the peri-ependymal tissue. The considerable enlargement of the canal, termed "syringomyelia," is generally associated with a peculiar growth in this position, probably congenital in origin; the condition is described in another chapter. In other forms of tumour, cysts occasionally develop, and hæmorrhage may occur into such cysts, or into the softened tissue in the vicinity of the tumour, or even in the

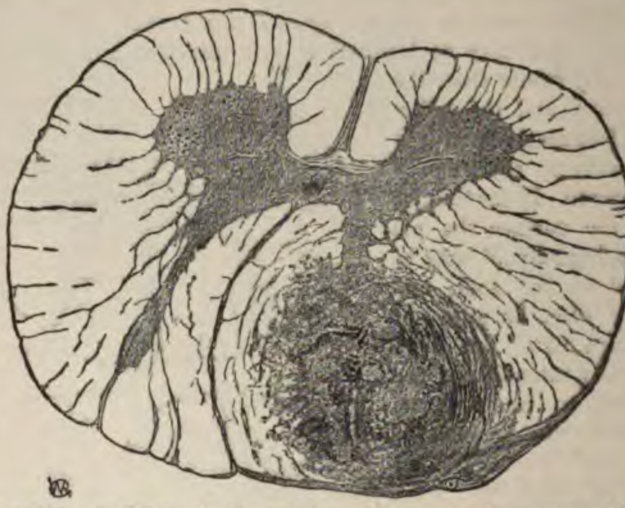

FIG. 182.—Syphiloma in the spinal cord, occupying the position of the posterior cornu and adjacent parts of the posterior and lateral columns, in the lower cervical region. The tumour presented the usual combination of grey translucent, and yellow cheesy areas. A second similar, but much smaller tumour existed higher up. The symptoms were complicated by hemiplegia, &c., of cerebral origin, due to a gumma in the brain. Paralysis with rigid flexor contracture of the left arm and leg were apparently due to the growth. The disease was certainly syphilitic, but it is noteworthy that it had developed during continuous treatment by full doses of iodide, which had entirely removed earlier symptoms, but to which the system had apparently become accustomed.

substance of a soft growth itself, especially when this is a glioma. From the region of the growth, secondary degenerations can frequently be traced upwards and downwards, but these are often slighter than the apparent damage would suggest, sometimes by reason of the remarkable tolerance of the nerve-fibres to pressure if this is slowly developed, sometimes because a rapidly growing tumour has not given time for them to become conspicuous. As regards vertical position, growths may occur in any part, but are said to be most frequent beneath the lower cervical spines, and in the upper and lower dorsal regions, because (it is conjectured) there is least room at these places, although why the result should follow from the cause is not clear.

SYMPTOMS.—The symptoms produced by tumours within the spinal canal, whether these are within the cord or outside it, resemble in their general characters those which we have already considered as resulting from growth and caries in the bones of the vertebral column. The resemblance is most close in the case of extra-dural growths, and least so in that of tumours within the substance of the cord. The chief differences are the relative preponderance of symptoms of early irritation of the cord itself when the growth is within it, and the frequent indications of a transverse extension of the damage from one side of the cord to the other.

In the majority of cases pain is prominent through the whole course of the disease. It is usually the earliest effect, severe both before and after the development of other symptoms. The pain may be intense along the course of the nerves which arise at the level of the tumour, and also in the parts supplied by nerves below that level, *e. g.* in the legs, but it is not felt above the growth. It may be a sharp acute pain, but has often a "burning" character, and is sometimes "stabbing" or "rending." Dull aching pain may be felt between the attacks of severe pain, or before them, and may then give rise to the most frequent of all diagnostic errors—mistaking grave organic disease for simple rheumatism, on account of the similarity in the character of the pains. The intensity of the severe pain is usually very great, and it has more than once led the unhappy sufferer to attempt suicide. The pains are often felt on one side first, in one arm, in one side of the trunk at a given level, or in one leg, and a considerable time may elapse before they extend to the other side. Occasionally the pain is bilateral from the first. Dr. Starr has drawn attention to the rarity of tenderness of the nerves in the line of the pain.* It is generally greatest when the tumour compresses the cord from behind forwards, or *vice versâ* (Horsley); this is open to more than one explanation. Pain is sometimes increased by movement, but rarely in the intense degree common in tumours of the bone. When the growth is in the lower lumbar region, the pain is often first referred to the soles, and seems to ascend. The sensory loss, due to subsequent nerve destruction, follows a similar course. Pain is sometimes felt in the spine, frequently when the growth begins in the dura mater, rarely when it is within the substance of the cord. Tenderness of the spine often exists in the former cases, but is on the whole not common. Other subjective sensations may occur with the pain or in the intervals—"numbness," tingling, formication, &c., and they give significance to the pains as an indication of organic damage to the nerve-structures. The root-pains in the trunk are often accompanied by a sense of constriction, which may be very distressing. Hyperæsthesia of the skin frequently accompanies pain felt at the level of the tumour, less commonly that in the parts below. Very rarely pain is absent; chiefly in extra-dural lipoma, probably from the nature of the growth.

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* Allen Starr, 'Amer. Journ. Med. Sc.,' 1895.

Muscular spasm is another common symptom, most pronounced when the tumour springs from the membranes. There may be some rigidity of the back opposite the seat of the growth, usually associated with local pain. It is most marked when the disease is at the more mobile parts of the spine, especially when it is in the cervical region. In such cases the rigidity may be painful, and the pain may be increased by movement. Severe spasm in the abdominal muscles is often associated with severe girdle-pain. Contractures may develop in the limbs, both in those supplied by the nerves which arise at the level of the tumour, and are directly irritated by it, and also, although less commonly, in the parts supplied from the spinal cord below the level of the growth. Thus the tumour in one half of the cord in the cervical region (shown in Fig. 182) caused persistent flexor contracture in the arm and leg on the side of the growth, and in the early stage of dorsal tumours one leg only may be rigid, or more so than the other. The seat of rigidity thus needs to be specially noted, as well as its character, if we are to obtain from it diagnostic guidance, because it may be due to the irritation either of the nerve-roots or of the conducting tracts. Both forms sometimes occur before loss of power, but pain generally precedes them.

Paralysis, of gradual onset, is almost invariably one of the symptoms. Paraplegia is the most common form, but all four limbs may be paralysed by a growth in the cervical region. Very commonly one leg becomes weakened before the other, and occasionally one arm and leg suffer before those of the other side. Tumours which are situated in the middle line or in the centre of the cord, however, usually affect both sides at the same time, and the effects of a congenital growth may thus accompany and complicate those of an associated syringomyelia (*q. v.*). The proximal parts of the limbs seem, as a rule, to be first affected, although exceptions are occasionally met with.

The palsy, gradual in onset, is usually also slow in its extension, the rate of its increase depending on the rate at which the tumour grows and compresses the spinal cord. This is the characteristic course of the paralysis produced by a tumour. But in a large number of cases the loss of power comes on in a subacute, or even an acute manner, in consequence of the myelitis excited by the compression, which in such cases may run a course of independent rapidity. When the inflammation is thus in excess of its cause, the palsy may lessen as damage from the inflammation subsides to the degree of the damage from pressure. The symptoms that depend on compression slowly but surely increase as the tumour grows, and usually attain a high degree of intensity; the loss of power in the legs, for instance, becoming absolute and remaining so.

Loss of sensation may come on with the loss of motor power; more often after this has become considerable. It may be slight or partial at first, occasionally for a long time, but tends to become complete as the damage to the cord increases, and it often ascends the legs. It corresponds in distribution to the motor palsy when the disease is below the middle of the dorsal region; but if situated higher, and on one side, the sensory loss is often greater on the side opposite to the motor paralysis. Tumours of the cord furnish a considerable proportion of the cases in which the crossed symptoms of a unilateral lesion are well marked (see p. 272). In addition to the loss of sensation which results from the damage to the cord itself, areas of anæsthesia may exist in the region supplied by the nerves which are damaged by the growth, and in which the severe radiating pains are felt. If there is crossed sensory and motor palsy, these root symptoms exist, chiefly on the side opposite to the anæsthesia in the limbs. Inco-ordination of movement is not a common symptom of spinal tumours, but has been occasionally met with in cases of growth invading the posterior columns, and also as a result of multiple tumours of the nerve-roots (see p. 478). Loss of power usually co-exists and quickly becomes predominant when the growth is in the cord.

The condition of reflex action depends on the position of the tumour. In the lumbar enlargement or the cauda equina a growth abolishes reflex action in the legs, and in any position the trunk-reflexes are lost at the level of the lesion. But when the disease is situated higher up, reflex action is increased, and the excess of cutaneous reflex action is a very marked and early symptom of the disease; it is lessened and lost only when the lower part of the cord is damaged by secondary myelitis (superadded to descending degeneration and distinct from it). The myotatic irritability presents also the increase that invariably results from damage to the pyramidal tracts, and the usual tonic spasm also gradually develops and often reaches a high degree. Its loss, with muscular relaxation, sometimes met with under these conditions, depends on a descending myelitis, sometimes slight and partial.* Muscular contracture is superadded more frequently in tumours than in many other diseases, and rigid spasm in the flexors of the knee and hip may keep the heels in contact with the nates.

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* As was found to be the case by Francotte ('Fibrome de la Dure-mère spinale,' Liège, 1888) in a case of tumour compressing the upper dorsal region. Slight

Wasting of the muscles is for the most part confined to the regions supplied by the damaged nerve-roots. In tumours of the lumbar enlargement and cauda equina the atrophy of the muscles of the legs is a very conspicuous symptom. Considerable muscular wasting in the legs may result from an irritable growth in the cord above the lumbar enlargement, even in the cervical region, (but such atrophy does not reach the extreme degree produced by growths in the lumbar enlargement or cauda equina,) and this condition is further distinguished by the preservation of the muscle-reflex actions (knee-jerk, &c.), and often by their excess (foot-clonus). Vaso-motor disturbance sometimes occurs in the early stage of the disease, chiefly in the distribution of irritated roots, where flushing of the skin has been noted, and the so-called "meningitic streak" ("*tache spinale*") may often be produced. Occasionally vaso-motor œdema is a conspicuous early symptom. In the later stages bedsores are very common and often severe. Dilatation or contraction of the pupil is common when a tumour is situated in the lower cervical region. Loss of control over the sphincters usually accompanies motor palsy in the legs; their state corresponds with that of reflex action (p. 267).

The course of the symptoms is variable, and depends on the rapidity of growth of the tumour, and on the share which secondary myelitis takes in the production of the symptoms. As a rule the root symptoms occur first, and in growths outside the cord they may exist alone for months, and even, in the case of a slowly growing tumour, for years before the symptoms of damage to the cord itself are added. On the other hand, they may be entirely absent in growths which commence within the cord itself. In some cases the symptoms are steadily progressive from first to last. In others the progress is intermittent; periods of increase in the symptoms alternate with stationary periods. Their course is variable, even in cases in which the seat of the tumour and its nature are the same, because they depend on the secondary changes in the nerve-elements, and the course of these (such as the myelitis induced by pressure) is subject to variations, the cause of which is often indistinct. Thus the chronic course of the symptoms is often varied by periods of rapid and even sudden increase, especially in the case of growths that involve the spinal cord itself; and symptoms that have thus developed with rapidity are apt (as already mentioned) to subside, if not completely, at least in considerable degree. Hence a rapid increase of the cord symptoms may be followed by positive improvement for a time, but scarcely ever by an

descending inflammation of the right anterior cornu coincided with flaccid palsy of the right leg. It should have been mentioned that the connection of a similar loss of myotatic irritability with descending inflammation in dorsal myelitis (referred to at pp. 264, 271 and 360) has been proved in two other cases by Francotte (loc. cit., p. 9).

actual disappearance of the manifestations of the disease. The most important element in the course of the disease is the tendency to a lateral extension of the symptoms, the expression of the transverse extension of the damage, as the several structures of the cord, at the same level, successively suffer. Although there is sometimes an extension downwards of the interference with the central functions of the cord, when secondary myelitis descends to the lumbar enlargement, there is scarcely ever an extension upwards above the level of the initial interference with function. A very small, slowly growing tumour, even within the cord, sometimes causes no symptoms.

The chief differences between the symptoms of tumours of the cord and membranes, besides those mentioned on p. 615, are that, in the latter, the root symptoms are more often severe, and that the cord symptoms occur later, and are at first more limited in extent. Spinal pain, tenderness, and rigidity are more common. In cord tumours the radiating pains, due to irritation of the nerve-roots, are more often trifling or absent, unless the growth begins in the neighbourhood of the posterior horn; then they may be pronounced. The cord symptoms are often bilateral from the first, and in central tumours they may develop equally on the two sides. If they are unilateral at the onset they extend to the other side sooner than in the case of meningeal growths. In the case of tubercular growths the symptoms of irritation are usually subordinate to those of impaired conduction, and pain is felt chiefly in the legs (Herter). Extensive muscular atrophy is more common in tumours of the cord than in those of the membranes (except in the case of tumours of the cauda equina), because central tumours may damage the grey matter through a considerable vertical extent, and are most common in the enlargements, and also because myelitis spreads in the grey matter far more frequently when the exciting growth is in the cord itself, than when it is in the membranes.

In tumours of the cervical region, the interference with movement of the neck, from the rigidity of the muscles, is often well marked, especially when the tumour is in the membranes. The pains due to irritation of the nerves are felt in the arms, and are often associated with muscular atrophy. The excess of reflex action in the legs is very great, and if the influence of the tumour is chiefly on one side, hemiplegic weakness, with crossed anæsthesia, is present.

Growths in the dorsal region of the cord usually cause severe radiating pains in the trunk, often accompanied by an intensely painful sense of constriction, by hyperæsthesia, and sometimes by anæsthesia. The simple, merely sloping course of the dorsal nerves reveals the precise position of the disease more clearly than do the symptoms in the arms when the tumour is in the cervical region. The interference with the trunk-reflexes often corroborates the indications afforded by the subjective symptoms. Spinal tenderness may be also present, but rigidity of the spinal muscles is not common. Reflex action in the legs presents the same excess that results from tumours in the cervical region, but sometimes, especially when the growth is near the lumbar enlargement, or the pressure myelitis has been acute, the descending myelitis, as we have seen, may abolish the reflex action and remove spasm, if this previously existed.

The symptoms produced by tumours which involve the lumbar enlargement vary considerably according to the precise position of the growth. The characteristic pains are felt in the legs, and the muscles supplied from the part invaded or compressed undergo atrophy, and present loss of reflex action and of myotatic irritability; but the anæsthesia is often limited in area. A growth in the upper part of the enlargement may cause wasting only in the flexors of the hip and extensors of the knee, with loss of the knee-jerk and anæsthesia in the front of the thigh, while the muscles below the knee present simple palsy with myotatic excess. Thus a man, who had had syphilis, complained of pain in the left groin and thigh, and presented wasting of the extensors of the knee, with anæsthesia in the front of the thigh; there was loss of the left knee-jerk, and a foot-clonus on each side. He recovered partially, but there was doubtless a small syphiloma at the upper part of the lumbar enlargement on the left side, interrupting the reflex arc, damaging the left motor and sensory roots, and compressing both pyramidal tracts. A growth in the membranes adjacent to the middle or lower part of the lumbar enlargement usually causes symptoms of wide distribution, affecting the whole of both legs, and the early symptoms of irritation often involve the distribution of the upper lumbar nerves. But a growth at the same level within the cord may affect chiefly the muscles of the lower leg, and cause anæsthesia in the region supplied from the sacral plexus. There may also be palsy and wasting of all the muscles of both legs, rom extensive secondary myelitis, but even then the sensory loss is usually limited. In lumbar tumours there is early palsy of the bladder and sphincters. Examination of the sphincter ani shows absolute paralysis, and not the pure reflex action present when the disease, arresting voluntary influences, is higher up the cord. The tendency to bedsores is commonly strong in tumours of this part.

Growths situated in the cauda equina, which are below, and do not affect, the cord itself, produce symptoms very similar to those due to tumours of the lumbar enlargement, but the symptoms are bilateral from the first in the majority of cases. Rarely one leg is affected alone at the outset. The symptoms usually begin in the lowest parts of the legs, which are throughout involved in greater degree than the upper parts, and the flexors of the hip may escape altogether. Muscular contraction is much less common than in tumours situated higher up. The anæsthesia is chiefly in the region supplied by the sacral plexus; the pains, usually very severe, are first felt in the same region, and there may be severe pain on each side of the sacrum radiating to the region of the sciatic nerves. The muscles below the knee rapidly waste, and often (but not always) present the degenerative reaction. These symptoms were well marked and characteristic in the case figured and described at p. 612 (Fig. 179), and the condition of the muscles suggests that the absence of any voltaic irritability in the muscular fibres was due to their early and intense fatty degeneration. In many cases the symptoms are irregular in distribution, the nerves are included in the growth, and are often damaged unequally, sometimes less than might be expected from the size of the tumour, which may be considerable before its expansion is arrested by contact with the bony walls of the canal. Hence the loss of sensation may be partial, and reflex action may not be completely abolished. In the case above mentioned, for instance, a prick on the sole caused a reflex contraction of the muscles in the upper parts of the legs, but of these alone. In many cases, however, the loss of sensation has been absolute, and reflex action also has been entirely lost. The tendency to trophic changes is great, and an affection of the sphincters is almost invariable. Cystitis and its consequences readily occur, and may reach a high degree, in consequence of residual accumulation, when the patient is conscious only of slight difficulty of micturition.

Multiple tumours (met with especially in cases of tubercle and gliomatosis) may give rise to very complex symptoms. If there are only two tumours, they may be indicated by the successive development of focal symptoms, similar in character but different in seat. In some cases in which there are many growths, only one or two of the largest have caused symptoms, and the smaller tumours have run a latent course. In other cases in which many tumours have caused slight symptoms, these have simulated closely a system-disease of the cord. Thus, in the remarkable case recorded by Hughes Bennett, multiple growths on the posterior nerve-roots produced the characteristic manifestations of locomotor ataxy.* When cerebral tumours have co-existed with multiple spinal growths, the symptoms of the latter have usually been lost in those of the intra-cranial disease which has often preceded the spinal lesion.

The duration of the symptoms of tumours of the cord and membranes, those of syphilitic nature being excluded, varies from three months to five, or even ten years. Equally variable, but shorter, is the time that elapses before the symptoms reach their full development, which depends partly on the secondary inflammation, partly on the rate of growth of the tumour. From the former cause, the time may be only a few days, when the myelitis is acute; from the latter it may be as short as three or four weeks, in tumours of tubercular or syphilitic nature. In the majority of cases death ensues at the end of from one to three years from the commencement of the symptoms, but these last longer when the growth is outside the dura mater than when inside the sheath. The immediate cause of death is usually the direct or indirect effect of the tumour; but in syphilis death is sometimes due to disease elsewhere, and in tubercle, frequently to general pulmonary tuberculosis (which usually precedes the spinal growth), or sometimes to meningitis.

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* 'Clin. Trans.,' 1885.

Diagnosis.—The evidence of a growth consists in the symptoms of a focal lesion of slow development, and especially in the presence of signs of compression and irritation (pain and rigidity), while the significance of these is increased by indications that the disease began at the surface of the cord, and sometimes by the history of a cause, as syphilis. Although no symptom is actually pathognomonic, yet the combination of the symptoms, and their course, are in most cases sufficiently characteristic. Those of greatest diagnostic importance are, first, the pain that is felt in the parts at the level of the tumour, or below it, and sometimes in the spine; secondly, the progressive paralysis, motor and sensory. Of less but still considerable importance are the rigidity of the muscles of the spine, the muscular contractions in the limbs, and the early and marked excess of reflex action when the cord itself is involved. In the course of the disease the most important features are the steady progress of the symptoms, and especially their commencement on one side and extension to the other. Of causal indications that occasionally assist the diagnosis, the most important are a history of constitutional syphilis, the presence of tubercular disease elsewhere, and, in rare instances, the existence in other parts of such multiple growths as are known to occur also within the spinal canal, especially multiple sarcomata and neuromata.

The diagnosis of multiple growths depends on the recognition of the successive development of the characteristic symptoms in more than one focus. It is rare for more than two of many tumours to cause definite symptoms. The greatest absolute difficulty in the diagnosis of a spinal tumour is presented by the cases sometimes met with, in which an intra-cranial tumour precedes the growth within the vertebral column, and the symptoms of the latter are overlooked in the presence of the severe manifestations of the former. In most cases, however, the additional symptoms, which usually exist, may be detected by careful observation, and their significance recognised. Growths involving the cauda equina cause symptoms like those of tumours in the lumbar enlargement, save that the effects are from the first bilateral in even more pronounced degree, and the evidence of interference with the nerves (loss of reflex action, nerve pain, &c.) is even more conspicuous.

The differential diagnosis involves the distinction from diseases of the vertebral bones, of the membranes, and of the cord itself. Caries of the spine has many symptoms in common with tumour, but the root pains are rarely very severe, and the effects of compression of the cord are usually bilateral, either from the first or very soon after their commencement; the paralysis seldom reaches a considerable degree in one leg before the other suffers. The signs of bone disease are rarely long absent, and repeated examination usually reveals tenderness and irregularity of the vertebral spines. From growths in the bones of the spinal column the diagnosis may at first be impossible. But the pain of tumour is rarely increased by movement to the same intense degree as when the growth commences in the bone, unless the tumour is in the cervical region of the cord, and then the greater accessibility of the vertebral column renders the exclusion of bone growths comparatively easy by local examination. In the dorsal region a growth in the bone may long escape detection, but even here the influence of movement on the pain is often extreme, far greater than in the case of tumours within the canal. The effect of the movement of the vertebræ on the nerve-roots, when compressed as they pass through the foramina, is far greater than when they are compressed within the canal. Sooner or later a local enlargement may present itself externally, and show the nature of the case.

"Hypertrophic pachymeningitis," affecting the cervical region, is the only meningeal disease which closely simulates the symptoms of tumour, but its effects are usually from the first bilateral, and they have a considerable vertical extent. Hence it is only with central tumours, which grow through several inches or more of the cord, that the disease is likely to be confounded. In each malady there may be muscular atrophy in the arms, and paralysis, without wasting, in the legs. But in central tumours the pains in the arms are usually much slighter than in the thickening of the membrane, and there is rarely the early and limited impairment of all forms of sensation which results from the damage to the nerve-roots in the latter disease. This is also true of syringomyelia, in which tactile sensibility usually persists, although untypical cases may present much difficulty. The diagnosis is considered more fully in the account of that disease.

The affection of the cord itself which most closely simulates the symptoms of tumour is a very chronic transverse myelitis. In such a case as that shown in Fig. 108, p. 383, the diagnosis may be a matter of extreme difficulty. The radiating pains in this case were severe and local, and the limitations of the symptoms, for a long time, to one side, with crossed sensory and motor palsy, presented an almost perfect analogy to the effects of a growth. But it is very rare for severe and acute radiating pains to result from focal myelitis, although a painful sense of constriction is common; moreover, in tumour, one half of the cord seldom suffers severely without the functions of the other half also being very soon impaired in some degree. The limitation of chronic myelitis to a single focus is also very unusual; a considerable vertical extent of the cord is usually involved in varying degree. Hence the practical difficulty in diagnosis is not so great as, at first sight, might appear. Acute or subacute myelitis can only be confounded with a growth in cases in which inflammation results from pressure, and develops with independent energy and rapidity. In such cases the occurrence of myelitis must be recognised, and the diagnostic problem is the detection of the preceding symptoms of the

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many cases. If the patient has had constitutional syphilis the syphilitic nature of the growth is highly probable, and the probability is further raised in degree if the growth developed rapidly, so that the symptoms attained a considerable intensity in less than three months from their onset. The co-existence of a cerebral lesion also increases the likelihood of the syphilitic nature of the growth. Either of the last two considerations suggests the same conclusion in any case in which syphilis, although not proved, is possible; but it must be remembered that cerebral and spinal tumours, of other than syphilitic nature, may co-exist. In the same way, tubercular and scrofulous processes suggest the tubercular nature of a tumour. Most cases of tubercular growths have occurred in adults, the subjects of phthisis. The diagnosis is made more probable by rapidity of course, by the speedy extension of the symptoms to the second side, by the slightness of evidence of irritation, and, in some cases, by periods of arrest of the progress of symptoms that were slow in their development. Such arrest indicates an arrest of the growth of the tumour, which we cannot infer from the subsidence of symptoms that developed rapidly, and may have been due to secondary inflammation. When a rapid onset is due to this cause, irritation symptoms are usually pronounced.* Tumours elsewhere always constitute strong evidence of the nature of one in the cord, but such an indication is rarely available, since the spinal canal is rarely the seat of secondary growths. Hydatids can only be diagnosed if others are present where their nature can be recognised; this has hardly ever been the case in the rare instances in which such tumours have existed within the spinal cord. Multiple tumours are probably either tubercle, sarcoma or neuroma. In the absence of these indications a meningeal tumour is probably a sarcoma, perhaps fibrous or myxomatous, and one within the cord is probably a glioma or tubercular, because these are the most frequent growths in those situations. A diagnosis founded on mere frequency of occurrence has necessarily only a low degree of probability, and must be sometimes wrong; but it may be highly probable that the growth is one of two or three kinds, and this may be of considerable practical value. To the surgeon the point is of great importance.

PROGNOSIS.—Unless the tumour is syphilitic, or can be removed, the prognosis is necessarily most grave. Of all syphilitic lesions, growths are those that are most amenable to treatment, and the effects of which most certainly pass away if they have not reached an extreme degree or lasted too long a time. If, however, the damage has been allowed to remain unchecked for several months, recovery may be imperfect. Tumours of most other kinds steadily increase, and the resulting damage is on the whole steadily progressive, although its course may be varied by stages of rapid (myelitic) increase in the symptoms, followed by stationary periods and even by transient improvement. In the case of growths outside the cord the prognosis has been rendered much less gloomy by the progress of surgery.

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* Thus numbness in the left leg, with increased sensitiveness to pain in the thigh, was quickly followed by complete paraplegia; the hyperæsthesia became restricted to the region supplied by the first lumbar nerves. Death occurred two months after the onset of the symptoms, and a tubercular tumour was found in the upper part of the lumbar enlargement, occupying almost the whole thickness of the cord (Ludeck, 'Jahrb. d. Hamburg. Staatskr.,' 1896).

TREATMENT.—If the growth is syphilitic, appropriate treatment should be energetic and prompt, so as to effect some diminution of the pressure as quickly as possible, since the longer this continues the more considerable are the degenerative changes, and the longer these will endure. Even the influence of a few days before treatment becomes effective may make a difference of weeks in the duration of symptoms, and lessen the degree of ultimate recovery. Whenever the symptoms suggest a tumour, and syphilis is possible, it is most important that suitable treatment should be employed; it can do no harm if the tumour is not syphilitic, and may save the patient's life and strength if it is. Syphilis can only be excluded when there has been no *possibility* of infection. In many cases of late syphilitic lesions there is no history of secondary syphilis, and in others there is no history of a primary sore. Hence it is *a priori* certain that in some cases of late lesions a history of both primary sore and secondary symptoms will be absent, and, as a matter of fact, such cases are met with not unfrequently. Of course these cases cannot be counted as syphilitic in any scientific investigation into the influence of this disorder, but in treatment we have often to allow weight to considerations which are insufficient to influence scientific investigation, and treatment determined by the mere possibility of syphilis is often justified by its results. Tubercular tumours are also amenable to treatment to some extent, but they are too rare to be of much practical importance.

In most other cases we can do little more than treat symptoms, relieving pain by sedatives, watching the state of the bladder, preventing the occurrence of cystitis, and guarding the patient from bedsores. We must remember that sedatives will probably be needed for a long time, and they must therefore be used as sparingly as possible, lest custom and tolerance deprive them of their power. Cocaine may be used in many cases as an aid in economising the influence of morphia.

But many tumours within the spinal canal may be removed, at any rate if they are outside the substance of the cord. The anticipation that such growths would be found removable was expressed in the first edition of this book (1886), in words that may be quoted because they were soon afterwards realised:—"Modern methods render the opening of the spinal canal far less formidable than it formerly was, and the removal of a tumour from the membranes of the cord would involve less immediate danger of serious consequences than the removal of a tumour from the brain."

On the other hand, growths within the spinal cord are in a very different position with regard to surgical procedure, since the removal of such a tumour could hardly fail to cause a traumatic inflammation that would damage and perhaps destroy all the elements of the cord at the spot, and would probably be permanent in its effects, especially when these structures have already suffered from pressure. At the same time the early removal of a small growth might possibly be followed by the regeneration of conducting fibres that are on the other side of the organ, and the return of their function, lost only through the effects of pressure.

In a man with complete paraplegia, motor and sensory, of slow development, accompanied by attacks of agonising spasm, a diagnosis of tumour led me to advise the removal of the growth. This was done by Mr. Horsley, and the result has been the permanent recovery of the patient. Although the intense spastic paraplegia showed that there must have been complete degeneration of the pyramidal fibres, every indication of this has passed away. The tumour was a myxo-fibroma, about the size of a split almond, which had so compressed the cord in the upper dorsal region as to reduce its thickness to about one half. It was within the dural sheath, and adherent to the cord.* It is certain that many growths might be removed without difficulty, and with a secure result, although not many instances have been since recorded.

In connection with an operation, a question sometimes arises that is of very great importance, and often of equal difficulty. The symptoms are consistent with the syphilitic nature of the growth; and this cause and nature cannot be excluded, except by the disproof afforded by the inutility of treatment. But it is essential that this disproof should be afforded before an operation is decided on; and the question arises, how long should be allowed for this process of practical exclusion,—how long should treatment be continued without result to justify the last resort? It needs to be longer when the symptoms are of considerable duration than when they are recent, since secondary changes of long duration cannot be quickly removed. Time is needed in every instance to permit a trustworthy conclusion, but a month is probably always enough to enable a result to be reached that will justify either further delay on the one hand, or immediate procedure on the other.

Finally, one injunction given by Mr. Horsley may be emphatically endorsed. If it is clear that the growth is not syphilitic, and that no good can be done by other treatment, delay in an operation can only cause harm—can only result in a less favourable state for the proceed-

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* The details of the operation are full of instruction in reference to similar procedures, and are given in the account of the case in the 'Med.-Chir. Trans.,' vol. lxxi, 1888. The surgical aspect of the subject is too large to be entered on here.

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dilated central canal; but as there is no real difference between this and other varieties, the designation, being unnecessary and even misleading, is falling into disuse.

To understand the origin of these conditions it is necessary to remember the mode of development of the spinal cord. The sides of the primitive furrow of the embryo coalesce, so as to form a canal of relatively large size, the walls of which are thinner in front and behind than at the sides, and consist of elongated cells, some of which are arranged regularly at the inner surface of the cavity, like an epithelium. The anterior wall first becomes thickened, to form the white commissure and the front part of the grey, while an abundant growth of cells occurs at the sides of the tube, and extends forwards (*i. e.* downwards in the position of the embryo), thus forming the lateral and anterior columns; between these two projections is the depression of the anterior fissure. Next occurs a growth backwards of the now thick sides, which forms the posterior parts of the lateral columns and the adjacent part of the posterior columns, and with this the posterior roots are connected; this forms the "root-zone" of the posterior columns. The posterior median columns are formed later. The central canal extends backwards between these two rudimentary postero-external columns, being closed behind only by a thin layer of cells. As the posterior columns increase in thickness the posterior part of the canal becomes narrowed, and its walls unite near its anterior extremity so as to form the posterior grey commissure, and divide the canal into two parts. Of these the anterior becomes the permanent canal, while the posterior is reduced to a narrow fissure between the posterior columns. Ultimately this posterior narrow part becomes closed by a growth of cells which occurs from behind forwards, as the median parts of the posterior columns develop. All these parts consist first of embryonal cells, which afterwards undergo a transformation into the nerve-elements proper, and the order of transformation is the same as that in which the parts were formed, the posterior median columns latest. The cells of the grey matter are formed before the fibres of the white columns. Some of the embryonal elements undergo a different and slighter change. They may almost be said to persist, constituting the neuroglia, and the gelatinous grey substance continuous with it around the cord, and also massed at the posterior root fissure and around the central canal.

The central canal ultimately lies in the anterior part or middle of the grey commissure. It may persist through life as a cavity, circular in section, or as a slit, antero-posterior or transverse, and is lined with epithelium (Fig. 183). Around the canal there are

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the cases as follows:—I. Hydromyelus, which runs a latent course without recognisable symptoms. II *a*. Primary (central) gliosis, with or without hydromyelus: (1) without cavity formation; (2) with splits and cavity formation. II *b*. Central gliomatosis, with or without cavity formation.

usually many nuclear elements, especially on each side of it, and these frequently fill up the lumen of the canal, so that its position is indicated only by an oval mass of small cellular elements (Fig. 183, D). The obliteration of the canal often occurs quite early in life, and is no evidence of any morbid process. The canal may be found obliterated in one part of the cord, and patent in another (C, D, Fig. 183). The simple closure of the canal by nuclei does not usually cause any distension of the part which is above the closed portion, or, at any rate, not more than a very slight increase in size, so trifling that its significance is open to question.

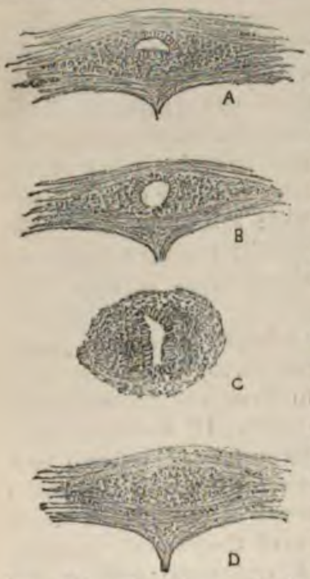

FIG. 183.—Central canal in normal cords. It has the form of a transverse slit in A, a vertical slit in C (from the conus medullaris), while in B it is circular. In each it is lined by columnar epithelium and surrounded by cellular elements, rounded, or angular from compression, mingled with granules. In D, which is from the same spinal cord as C, the position of the canal is occupied by a mass of nuclear tissue.

Syringomyelia occurs chiefly in the cervical and upper dorsal regions of the cord. The cavity is often closed below by gliomatous tissue, and the excess of this may extend up into the medulla and even pons. Occasionally the cavity exists through the whole length of the cord, and may extend into the medulla oblongata, and then affect the aqueduct of Sylvius and the tissue around it.

In the simple condition found in young children, or in adults when no secondary changes have occurred, the morbid change presents the two features already mentioned—a quantity of tissue of translucent aspect, which, from its structure, is clearly persistent embryonal tissue in which nerve-elements have not developed, and a cavity due to imperfect contraction of the canal. The change is always confined to the posterior half of the cord, and usually to the posterior columns, which, as we have seen, are formed last. It may, however, be present in one or both posterior horns, in which cavities also may be formed. The persistent embryonal tissue may be evidently increased by a process of various degrees of growth, and the cavity enlarged by distension. Further, when the persistent tissue is considerable in quantity it may break down, and thus give rise to a new cavity, or enlarge that which has developed from the primitive canal. Thus the cavity may be distinct from the canal, and in some cases the abnormal tissue around it is very small in amount, and reduced even to a mere wall. It may also be separate, because it is the hinder part of the embryonal canal, cut off by coalescence of the walls.

These conditions are shown very well in the adjoining figures, after Leyden (Figs. 184, 185). In the first, at A, the cavity presents nearly the shape which it has at one period of development, before the formation of the posterior columns is completed, and without the separation into two parts produced by the formation of the posterior commissure. It is bounded by a narrow layer of embryonal tissue, which widens out behind, having nearly the shape of the posterior median columns, which are, as we have seen, the last to be formed. In B and C the cavity is much larger, and the posterior columns are smaller, either by an earlier arrest of development or by greater distension, or, most probably, by the influence of both these agencies. The cavity extends up to the posterior surface of the cord, and is closed in there only by a narrow layer of cellular structure.

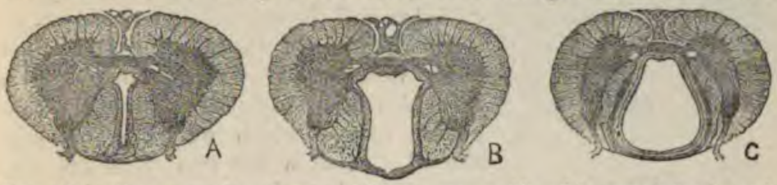

FIG. 184.—Syringomyelia, from a child two years old, with encephalocele and absence of cerebellum. (After Leyden.) In A, cervical region, the central canal is large, lined with epithelium, and from it a medial fissure extends backwards nearly to the periphery of the cord, limited by a layer of homogeneous tissue, more abundant at the posterior limit. In B, lower down, the fissure is enlarged to a cavity of considerable size, while in C it is still larger, and the posterior columns are reduced to a narrow zone between the layer of tissue which bounds the cavity and the grey substance.

In Fig. 185 we have somewhat more complex conditions. The cavity in the first section closely resembles in appearance that in A of the last figure, but differs in the important fact that the posterior commissure has apparently been formed and the central canal developed in the normal manner, although it has become obliterated by nuclei. This is clear if the commissure is compared in the several figures. The cavity, therefore, does not, as in Fig. 184, represent the whole of the primitive canal, but only the posterior portion after its division. The adjacent tissue is much more abundant than in the other case. The projection into the posterior extremity of the cavity indicates a process of active growth, which is also shown by the irregularity of the cavity in B, the large amount of tissue, and the wide separation of the posterior horns. In C, from the lower part of the cord, the embryonal tissue occupies two oval areas in the posterior column (by mistake, clear in the figure), while the cavity is closed or nearly so. (Lower down, a cavity existed in the centre of each of these areas, apparently due to breaking down of the tissue.) In D, again, the embryonal tissue occupies only the middle line, as a wedge-shaped area, due to the defective formation of the medial part of the posterior columns. It has been mentioned that the neuroglia may be regarded as a persistent, slightly modified embryonal tissue, and these exuberant masses of tissue bear considerable resemblance in structure to glioma, so that the condition has been called "gliomatosis." In each of the cases the brain participated in the defect development; there was internal hydrocephalus, and the cerebellum was absent.

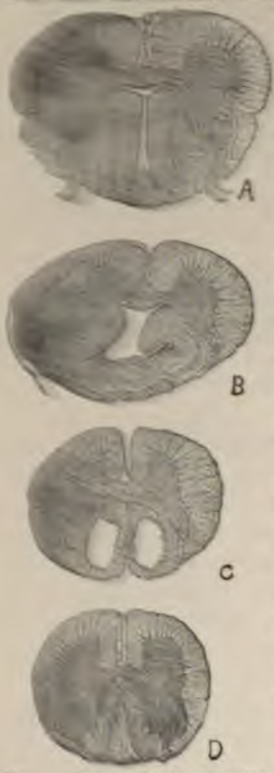

FIG. 185. — Syringomyelia, from a child aged two and a half years, with internal hydrocephalus and absence of cerebellum. (After Leyden, 'Virchow's Arch.,' Bd. lxviii.) A, B, cervical region; gelatinous (embryonal) tissue in the posterior columns encloses a medial cavity, lined in places with cylindrical epithelium, and believed (probably wrongly) to be the central canal. C, lower dorsal; in each posterior column is a mass of similar gelatinous substance (which, by a mistake, is shown in C as an open cavity). In D, lumbar region, this tissue occupies only the position of the posterior median columns.

The cavities in the adult cord present many varieties. The most common form resembles in its essential features the congenital disease which we have just considered. Between the two there are also differences, but the resemblance is sufficiently close, and the origin of the differences is sufficiently clear, to make it practically certain, as already mentioned, that the two forms are really the same, and that the form met with in adults is the congenital condition, persisting until later life. Of the characters which this form has in common with the congenital disease, the most important are the position of the cavities and the presence of tissue in their vicinity of embryonal or gliomatous nature. The differences depend on an apparent increase of this tissue by a process of growth, on the greater damage to the other parts of the cord by the distension of the cavity, and on the extent to which the formation of new cavities or enlargement of old ones takes place by a process of disintegration of the newly formed or persistent tissue. The damage to the cord produced by distension of the cavity involves most the nearer and less resisting parts. The grey substance, therefore, suffers in greatest degree; the white columns to an extent that depends in part on the precise position of the cavities, as presently to be described. The latter also present, in many cases, the complicating changes of secondary degeneration, ascending or descending, as shown in Fig. 186, due to the compression of certain tracts at some specially affected spot.

As the simplest condition, we may have a dilatation of the central canal surrounded by gliomatous tissue disposed as in congenital cases. This tissue is often more abundant, and forms a more distinct mass, lower down the cord, below the cavity. A good example of this condition in slight degree is presented by the cord, the seat of chronic myelitis, represented at Fig. 109, p. 385. In the cervical region (A) there is a large central canal, bounded by a thick layer of gliomatous tissue. In the inner part of this tissue, limiting the cavity, is a sinuous membrane, fibrous in structure. The origin of this membrane is not easy to explain, but it is often found in cavities of this character, and is evidence of the similar nature of those that differ much in other respects. In the lumbar region (B) the tissue, which bounds the cavity in the other section, forms a large round mass in the position of the canal, obliterating it.

When a central cavity appears not to be the canal itself, but to be situated behind, it is then apparently often due to a persistence of the fore-part of the posterior portion of the canal, after the posterior com-

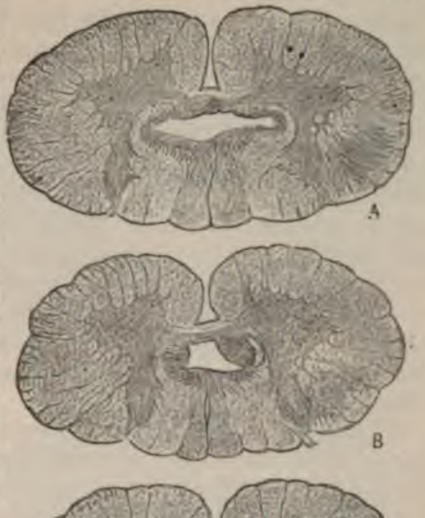

Fig. 186.—Syringomyelia, from a case of tumours of the pons and cauda equina, with some diffuse sclerosis in the dorsal region of the cord. A, B, and C are sections of the cervical enlargement. The large cavity in A is perhaps the dilated canal, since it is lined in front with epithelium, but in front of it, in the posterior commissure, is a group of nuclei like an obliterated canal. The zone of tissue around the cyst is composed of interlacing fibres and nuclei, and processes extend from it into the posterior columns both in front and behind. In B the cavity is smaller and the wall thicker, while in C the tissue about it forms a considerable mass, and a second small cavity has formed in the middle line, apparently by the breaking down of the tissue. In the dorsal region (D) there was merely an unusually large accumulation of nuclei in the position of the canal, which, in the lumbar region, had the normal aspect (E). The cord presents degeneration of the right pyramidal tract (descending from the tumour in the pons), and of the post. med. cols. and ant. lat. ascending tract, probably ascending from the sclerosis.

missure has been formed. In Fig. 186, for instance, we have a central cavity, but in the commissure in front of this is a mass of nuclei having the usual aspect of an obliterated canal.* The cavity is surrounded by a zone of tissue, which in A and B sends off a fringe of processes, especially backwards. This tissue is increased in the lower part of the cervical region (C). In B a small cavity is formed between the tissue and the grey matter by breaking down, and in C another exists in the middle line behind the growth, probably due to deficient closure of the original fissure, and increased by a destructive process. Still lower, the commissure resumes its normal appearance, but the collection of nuclei in the position of the canal is unusually large (D). In the lumbar region the canal is patent (E). The degeneration of the posterior median column and of the lateral columns is secondary.

We have seen that in the congenital cases we must recognise a process of growth of the persistent embryonal tissue to account for some of the conditions met with, and a like process is suggested by the central mass in Fig. 109, B. Hence it is not surprising that in many cases the growth should attain the dimensions, and assume the characters, of a positive tumour. In most cases the tumour has been central in position, and has had the structure of a glioma. It has occupied a large part of the area of the cord at a certain level.† In the chapter on the tumours of the cord it has been pointed out that sarcomata also may grow from the tissue around the central canal, as in the case shown in Fig. 181. Hence it is not surprising that the condition of syringomyelia, even if this is congenital in origin, should be frequently associated with definite tumours. The growths have often been multiple, apparently the result of a wide-spread tendency, and perhaps connected with a wide-spread persistence of embryonal tissue, of which remarkable instances are sometimes met with. In the case shown in Fig. 186 there was a tumour of the pons and also one of the cauda equina. The nature of the growths is uncertain, but they were probably either sarcoma or glioma. The same tendency to morbid growths is illustrated also by Fig. 187, which is similar in many respects to that just considered. In this case also there was a tumour of the pons and one of the cauda equina, and there was also a central growth in the dorsal region, occupying the greater part of the area of the cord, to which the ascending degenerations were secondary. In A there is a large central cavity which is probably not the central canal, since, as in the last figure, the position of this is marked by the oval group of nuclei in the anterior part of the grey commissure. The zone of tissue which bounds it is narrow, but a few processes are given off behind. In B, first dorsal, this tissue forms a compact mass, and the processes are numerous and very similar to those in the last figure. In C the commissure is reduced almost to normal conditions. In this cord we have also an instance of the fact that cavities may occur in other parts, very similar in their general characters to those that occur in the central region. In B a large cavity occupies the intermediate grey matter and posterior cornu on the right side, with some morbid tissue bounding it on the medial side. In C the cavity is smaller

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* This is the probable interpretation. At the same time it is possible that the nuclei have not this significance, and that it is really the central canal. It is often more difficult than might be imagined to say whether a cavity does or does not represent the central canal. The presence or absence of an epithelial lining has generally been taken as a criterion, but it is doubtful whether this has any significance. Around the whole of the original cavity the inner layer of cells is arranged as an epithelium, and if any part of it persists it is probable that epithelium will persist also. Moreover the epithelium often disappears from the wall of the dilated central canal itself; always where it is enlarged by breaking down of tissue.

† As in an interesting case described and figured by Riesinger, 'Virchow's Archiv,' Bd. xcviii.

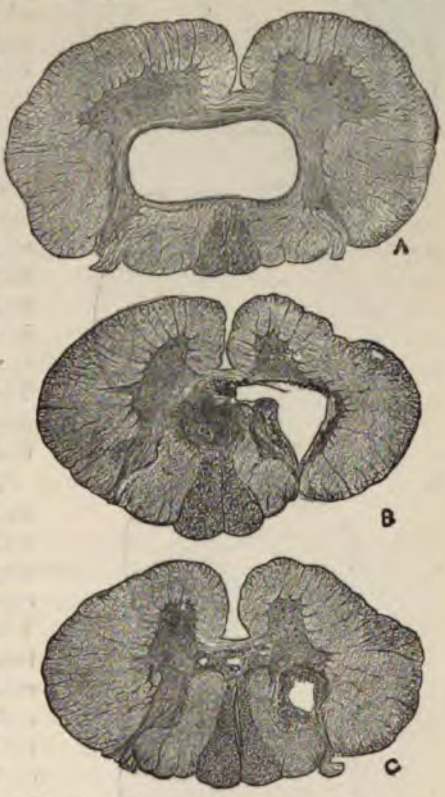

FIG. 187.—Syringomyelia, from a case in which there were tumours in the dorsal region of the cord, the cauda equina, and the pons Varolii. A, mid-cervical region. A large oval cavity lies behind the grey commissure, surrounded by a narrow wall, chiefly composed of fine fibres, but with a wavy membrane on the inner surface. In front of the posterior commissure, an oval group of nuclei has the aspect of an obliterated central canal. A few short processes extend from the wall of the cavity into the posterior columns. There is secondary ascending degeneration of the post. med. cols., right direct pyramidal tract, and ascending antero-lateral tract in all the sections. B, first dorsal. Behind the posterior commissure is a growth consisting of small cells, round and fusiform, the latter with round nuclei. It contains many vessels with thickened walls. A fringe of fine processes extends from it into the posterior columns. An irregular cavity occupies, on the right side, the posterior half of the grey matter, which is reduced to a narrow layer around the cyst. This is lined by a delicate nucleated membrane, outside which is a thicker membrane, lying in folds, the section of which is thus sinuous. Outside this again is a small-celled growth similar to that behind the commissure. In C, a little lower, this cavity is smaller, the sinuosities of the wall greater, and the growth outside it more abundant. (Still lower the area was entirely occupied by the growth.) In the posterior commissure there is only a quantity of loose nucleated tissue in the situation of the growth.*

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* For the opportunity of drawing these sections I am indebted to Dr. Dreschfeld. A full account of the case has been published by Dr. Harris in 'Brain,' Jan., 1886. The large growths were sarcomata.

and the tissue around it is more abundant, but easily distinguishable from the grey substance, while a little lower down the cavity disappeared and the tissue formed a compact rounded mass. The cavity is lined by a delicate layer of cellular membrane; outside this is a fibrous membrane, lying in folds which appear as sinuosities in the section. The existence of this membrane shows that the cavity is not simply formed by the breaking down of the tissue. The tissue is quite similar in structure to that which is behind the commissure in B.*

The posterior cornu is not an uncommon seat of cavities, which may extend through the whole length of the horn. An example of this is shown in Fig. 188. Hæmorrhage had occurred into the cervical region of the cord (A), which was enlarged and distended with blood, so that the precise character of the cavity in this region was indistinguishable; the blood also filled the cavity through the

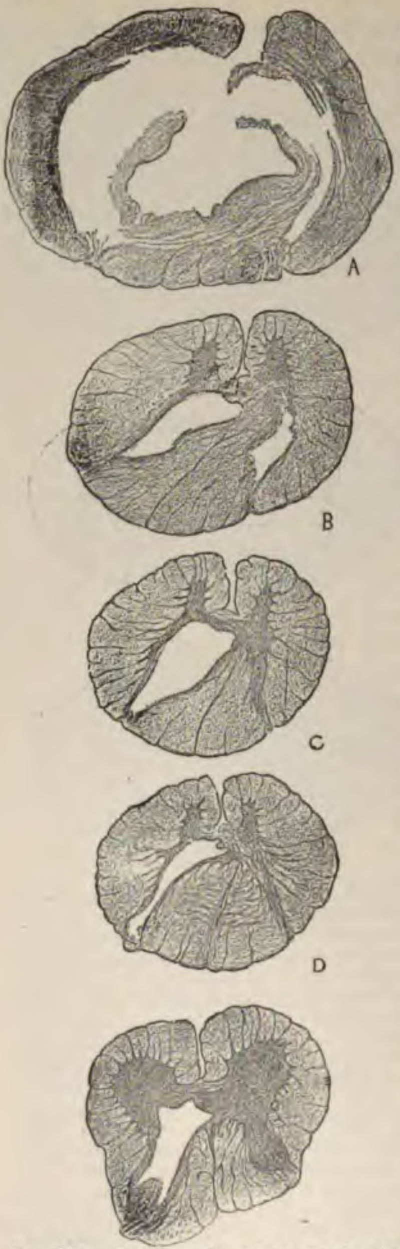

FIG. 188.—Sections of a spinal cord in which a cavity existed throughout its length. Hæmorrhage in the cervical region was the immediate cause of death. The extravasation had there distended the cord (A), and had burst into the cavity, which was filled with blood. A smaller cavity existed in the right cornu, in the upper dorsal region (B). In most parts the inner surface was slightly irregular, as if produced by breaking down of the tissue, but near the position of the central canal a sinuous membrane existed in places, without, however, any epithelial covering.

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* In connection with the tumour of the cauda equina in these two cases, it may be mentioned that the same coincidence has been observed in other instances. There was a tumour in that situation, for instance, in the case described by Riesinger, who rightly regarding the condition as congenital in origin, suggests that the tumour may be connected with the fact that the embryonal cord occupied the entire length of the canal.

entire length of the cord. In the upper dorsal region (B) there are two cavities, one in each posterior cornu, but that on the right ceased a little lower down, while the other extended to the lumbar region, as shown in the figure, reaching almost to the surface of the cord. The upper and inner extremity of this cavity is in the position of the central canal, no other trace of which exists. There is some abnormal tissue in the neighbourhood of the commissure, and also in C, where irregular tracts pass transversely from the median septum into the fore-part of the posterior columns. The presence of this tissue, and the relation to the central canal, suggest a congenital origin. During the reduction in size of the canal, in the development of the cord, the cavity presents at one period short lateral processes, and we can therefore understand how a still earlier arrest of development may lead to the persistence of a lateral process on one side or both, surrounded by residual embryonal tissue, and that the cavity should afterwards extend in this tissue or in the grey matter by a process of growth and disintegration, such as probably had occurred in Fig. 188, D. A comparison of D and E suggests that the two cavities were united in the middle line in the upper part of the cord, where the hæmorrhage took place.

In the conditions we have hitherto considered, definite limited regions of the cord have been involved through a considerable vertical extent. The changes are sometimes, however, much more diffuse in their distribution, as is shown by the remarkable lesions represented in Fig. 189. In this cord, which is that of an adult, we have a combination of the same two conditions of abnormal tissue and cavities. Scattered through the nerve-substance are tracts of a peculiar tissue, represented by the darker shading of the figure, and in many regions thus affected there are cavities, for the most part fissure-like, as if the cord had been split here and there. At first sight the appearance suggests an active and recent morbid process; nevertheless I think that the congenital origin of the condition is beyond doubt. It will be observed that the morbid changes, as in the other forms of syringomyelia, are chiefly in the posterior half of the cord; small areas of disease in the anterior columns, in the sections E and F, are the only exceptions. The minute structure of the abnormal tissue was exactly like that of the normal gelatinous layer beneath the pia mater. Besides the conspicuous areas figured, it could be traced here and there, in small tracts, through the whole of the posterior columns and the hinder parts of the lateral columns, and also in the posterior horns, in the grey matter of which it could be readily distinguished. The relation of the cavities to this substance is distinct; where they extend beyond it, this is apparently the result of the distension of the cavities and an actual fissuring of the cord, along the lines of connective tissue, by the pressure of the contained liquid. The medial cavity in C is doubtless due to defective closure of the original fissure between the posterior columns. In the lumbar region the nerve-fibres are absent from the front of the posterior columns, where a coarse network of this residual tissue encloses empty spaces.

The damage to the grey matter may entail alterations in the related nerves, and even, in cases of long duration, in those connected with the posterior roots. In the peripheral nerve-fibres of the skin and muscles, changes of the ordinary chronic degenerative character were found by Dejerine in cases in which sensation was altered or the muscles wasted.

Thus these various forms of syringomyelia, in spite of their variations in seat and form and general aspect, when closely studied, seem all to be connected with gliomatous tissue; and that the morbid state takes its origin in abnormal conditions during the development of the cord is practically certain from their seat and features. This lesson is clearly taught by the facts shown by the cases here illustrated. It is probable, therefore, that this conclusion is also true of cavities of similar character and position in which no gliomatous tissue can be recognised; and most of the hypotheses regarding the origin of such cavities from processes of simple myelitis and the like have no sufficient foundation. But we are still ignorant of the mechanism by which the defect in the process of development is brought about. It has been extensively ascribed to an inflammation of the embryonal neuroglia at an early stage of development, but of this there is no real evidence. It is, moreover, no explanation to assume a primary intra-uterine hyperplasia of the embryonal tissue. It is, indeed, possible that more than one morbid process during the early stage of development may cause the condition, and that closure of the canal below may have consequences during development which it has not at a later period. But it is important to recognise the fact that there is a gradation between the chief varieties, and that some are associated with developmental defects in the brain, to which they can only be related as the common expression of a defect in the developmental tendency of the germinal tissue; and what must be true of some forms is probably true of most. This may seem only to push the difficulty into the region of the inexplicable, but it is only placing the origin of the malady where we are compelled to refer the causation of so many congenital diseases, which, after all, bear but a small proportion to the physiological characteristics that spring from the same source. From these considerations it is probable that cases of syringomyelia are divisible into two classes: (1) those in which there is a congenital anomaly which may afterwards take on a process of overgrowth as distinguished from development, characterised clinically by very gradual onset of symptoms lasting, it may be, throughout several years; (2) those in which there is actual central new growth, frequently with cavity formation; and it may be associated with new growths elsewhere — glioma, sarcoma, syphiloma, — in which the course is a much more rapidly fatal one.

The symptoms of the disease have followed some illness, mental shock or anxiety, and traumatic influences, but it is probable that in no case have these done more than excite the manifestations of pre-existing disease, or induce local changes in the morbid state. The possible influence of injury has certainly been overestimated, since in some of the cases in which it seemed effective the malady was unquestionably congenital, and the traumatic influence only terminated its latent stage.[*]

Symptoms.—The manifestations of syringomyelia depend on the secondary processes of distension and growth, and the damage thus produced to the functional elements of the cord.[†]

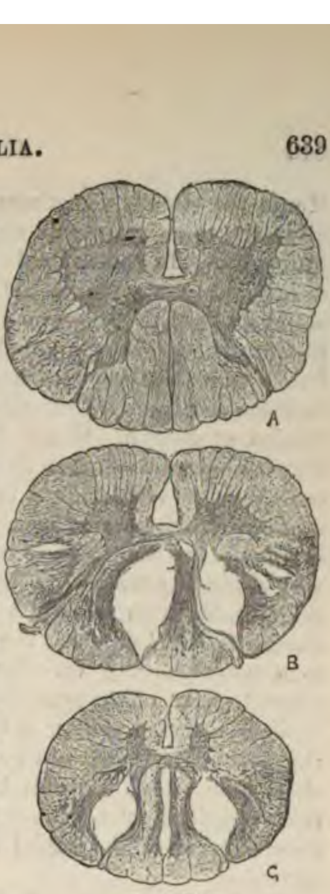

Fig. 189.—Cavities in the spinal cord. A, mid-cervical, presenting merely some increase of connective tissue in the left lateral column. In B there are several cavities having the aspect of distended fissures, the largest in the middle line of the posterior columns and the right posterior cornu; smaller ones in the lateral columns. Adjacent to each is a quantity of dense tissue, which had a peculiar and uniform structure. It consisted of a very fine network of fibres with minute nuclear bodies in the interstices. No larger cells were observed. Throughout the posterior and lateral columns this tissue could be seen, here and there, thickening the neuroglia, where no conspicuous masses were formed. It could also be traced in the posterior cornu, and was readily distinguished from the spongy substance by its slighter staining. The distribution of this tissue is shown in the other sections by the darker shading. In C the fissure in the middle line is narrow. In E and F the anterior part of the posterior columns contained a coarse network of this tissue, and no nerve-fibres. The central canal was obliterated, but the group of nuclei that marked its position could be traced throughout the cord.

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[*] Among the chief papers in which the subject will be found discussed are those of Schultze (see below); Hellich, 'Arch. Bohême d. Med.,' 1890, iii, No. 4; Kronthal, 'Neur. Centralb.,' 1888, p. 333; Oppenheim, 'Charité Annalen,' 1886, p. 409; Hoffmann and Schlesinger, loc. cit.

[†] They have been fully described by Schultze, 'Zeitschrift f. klin. Med.,' 1888, p. 525; and 'Verh. Cong. Inn. Med.,' 1886; and by Allen Starr, 'Am. Journ. Med. Science,' 1888, vol. xcv.

Hence the state is sometimes discovered after death, when it has given rise to no symptoms, and its existence has not been suspected during life. In young children, indeed, it appears never to cause any definite symptoms, while the disturbance of function is variable in occurrence and degree—much more so than in character. The central position of the disease entails considerable uniformity in the distribution of its local consequences, and hence also in the derangement of function that results from simple increase in the lesion either by distension or growth. Hence the symptoms are often such that they have considerable diagnostic importance, although in a minority of cases they are less uniform in character, probably because the disease is unsymmetrical, or irregular in position or extension, whether the symptoms are due to the distension of a cavity, or to this combined with adjacent growth of tissue. It then often causes symptoms indistinguishable from those of a tumour of neoplastic nature. Moreover the precise level at which the chief morbid process takes place leads to a corresponding variation in the position, and therefore in the general aspect, of the symptoms it produces. The fact that the lesion is almost always greater at the cervical region causes the arms to be the most common seat of its manifestations, while the occasional invasion of the fourth ventricle by the associated growth, and the occurrence of coincident secondary changes in this part, explain the involvement of some of the cranial nerves that has been met with in a few instances.

The symptoms are usually of slow development, increasing gradually in the course of years. Their main features are twofold, of which one is clearly and certainly intelligible, while the explanation of the other is a matter of hypothesis, although also not difficult to understand, at least in part, if the opinions expressed in the chapter on the functions of the cord are correct. The two leading features are loss of sensibility, chiefly to pain and temperature, and muscular atrophy, resembling in characters that met with in the "progressive" spinal form. Of the two, the sensory loss is the earlier and more constant. The muscular atrophy is a necessary consequence of the damage to the anterior cornua from the compression they endure, when and where the central cavity or adjacent growth attains such a size as to damage them seriously, and they may be reduced to a mere zone between the cavity and the outer ring of white substance. The destruction of the motor nerve-cells involves the degeneration of the nerves and the wasting of the muscles. The loss of sensation is intelligible if the path of painful impressions is by the posterior commissure.[*] The enlargement of the central canal and disease in its vicinity can hardly fail to damage early and much the fibres that cross at the level of the chief lesion, and constitute the path from posterior roots not very far below. The course of thermal sensations is still quite unknown; the only fact we have—the frequency with which such sensibility and that to pain are involved together—points to contiguity of path, and prevents surprise at the special affection of this form of sensibility in syringomyelia. Tactile sensations are lost only in rare cases; our ignorance of their path prevents us from explaining the fact, but we know, from other facts of disease, that it is distinct from that for pain. Moreover, the occasional localisation of the morbid process in the posterior columns, and even cornua, may explain the involvement of all forms of sensibility, and also its occasional restriction in area, since it may be due to direct interference with the posterior root-fibres after they have entered the cord.

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[*] See p. 224 as to a tract in the grey commissure described by Ciaglinski ('Neur. Cent.,' 1896).

In many cases the affection of sensation of pain and temperature is equal; in others the latter is lost to a greater degree and extent than the former. Its loss is often the earliest symptom, as the scars of burns may show. The loss to heat and cold may not correspond. Thermal or painful hyperæsthesia has preceded the loss, as with other forms of sensation when destroyed by processes that slowly damage the fibres, and irritate before they interrupt. Perversion of sensation has been noted, heat being felt as cold, and *vice versâ*. Spontaneous sensations are common, doubtless from the irritation of the fibres in the early stage, or of their upper portions when they are interrupted—feelings of heat or cold, and in some cases pains, various in character, sometimes paroxysmal like neuralgia, or sharp and brief like those of tabes, and occasionally referred to the joints, or felt chiefly in some part adjacent to the seat of the other symptoms. Pain in the spine may be complained of, and is, perhaps, a direct effect of the disease. Cramps also and shooting pains sometimes occur. Often the malady causes no suffering. The loss of sensation is usually sharply limited, and the arms or upper half of the trunk are its common seat. It is usually found in the same part as the muscular wasting, but is more extensive, especially below the seat of atrophy. Thus, when the muscles of the shoulders and upper arms only are wasted, the sensory loss may be in the forearms and hands. This we should expect from the fact that the crossing of the sensory path is some distance above its entrance into the cord, while the motor roots arise from nerve-cells mostly near their level of exit. Exceptions may be due to the affection of the root-fibres, as explained above. Further, the fact that the distension and damage always extend through a considerable vertical area of the cord makes the relation of the two sets of symptoms more extensive and less regular than if the disease were limited to a small region.

The "muscular sense" is said to have been generally normal when tested. Occasionally there has been unsteadiness on standing with the eyes closed.

The motor symptoms usually come on after the sensory loss, long after in slight cases, and consist in muscular weakness and wasting in the upper limbs; while, if the legs suffer, it is generally from simple spastic paralysis, such as would result from, and is no doubt due compression of the pyramidal tracts by the disease in the upper p of the cord. It is rarely complete. The unsteadiness of mo ment above mentioned we may connect with the tendency of t lesion to involve the posterior columns, and so probably to inte fere with the path from the muscles to the cerebellum. Rarely the is atrophy of the legs similar to that of the arms, and in such cas the disease extends into the lumbar enlargement. Reflex action the legs varies according to their state; it may be normal or increased the knee-jerks are often augmented, with rectus- and foot-clonus, or in the cases last mentioned, myotatic irritability may be lost—very rarely on one side only. A curious tremor of the limbs has been noted in some cases.

The muscular atrophy in the arms begins in and chiefly affects certain parts, according to the position of the greatest damage to the grey matter of the cord. It may be in the hand and region of the ulnar nerve, or in the muscles supplied by the musculo-spiral and radial nerves, or in the shoulder and upper arm muscles.* It is a slow wasting, with gradual lowering of the electrical irritability, as in progressive muscular atrophy, although, as in that disease, some groups of muscles may present indications of the reaction of degeneration in the early stage of their affection. Fibrillation is common. As the wasting and weakness increase in the part first affected, they spread to others, to the rest of the limb, and to the adjacent parts of the trunk. Weakness of the trunk muscles often leads to lateral curvature of the spine; and the convexity, owing to posture, is usually to the left. The occurrence of similar symptoms in the legs is extremely rare. Both sides usually suffer together; a unilateral affection has indeed been observed, but is altogether exceptional. The sphincters may escape or be involved.

Trophic disturbances in the extremities affected with sensory loss are not uncommon. The skin may become thin and glossy, or thick and horny. Acute changes may occur—eczema, herpes, or bullæ; whitlows; deep, obstinate ulceration, and even gangrene. More frequent, however, is simple vaso-motor disturbance, coldness and lividity of the extremities, or redness with swelling and heat, and sometimes a tough œdema, which may be local like a tumour, and afterwards pass away. The nails share the trophic changes in the hands, becoming grooved, fissured, and cracked; they may even drop off. The secretion of sweat is lessened in the part, as was well marked in one unilateral case,† or it may be increased. The action of pilocarpine has been found to be delayed, although ultimately increased (Grasset). The bones may become thickened or brittle, and joint changes (Charcot's joint), like those of tabes, sometimes occur.[*]

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* Blocq thinks that spastic paraplegia is chiefly met with in the cases in which the atrophy begins in the region of the ulnar nerve, and tabetic symptoms in the legs when it begins in the radial area; but the data for the generalisation need to be more extensive.

† Allen Starr, 'Am. Journ. Med. Sc.,' May, 1888; Rumpf, 'Neur. Cent.,' 1889, p. 257.

It is not common for the symptoms to extend into the region of the cranial nerves, although in occasional cases various disturbances of function have occurred, presumably due to the extension of the changes into the medulla and pons, and chiefly met with when there is upward extension of the associated growth, already mentioned. Thus there have been paralysis of one vocal cord, or of both, of the tongue, the palate, and of the face, difficulty in swallowing, disorder of respiration and of the heart's action. Taste and smell may be affected, and there may be sensory impairment in the face. Certain disorders may occur in the eyes, probably due to disease of the path of the sympathetic in the cord, or to that of the fourth ventricle. Thus the pupils are often unequal, the smaller being on the side of the more severe cord symptoms, on which the fibres for the radiating muscle of the iris are likely to be damaged. Narrowing of the palpebral fissure and slight ptosis have been also noted. Nystagmus is not rare.[†]

The course of syringomyelia is, as a rule, slow, and measured by years, although an apparent exception is presented by the cases in which it is associated with spreading overgrowth of the adjacent gliomatous tissue, or in which acute destructive processes occur in this and spread to its vicinity. In such cases a considerable development of the disease may be attained before symptoms are produced, and these may rapidly reach a considerable degree, and cause death in a few months or even less. In most cases death is the result of exhaustion, of the impairment of the functions of the medulla, or, more commonly, of some complication—bedsores, cystitis, and the like.

Diagnosis.—The resemblance of the disease to more common maladies led to its confusion with them during life, until the careful observations of the last few years have established features by which its existence can often be at least surmised. The most important characteristic is the combination of muscular atrophy with still earlier sensory loss, involving temperature and pain far more than tactile sensibility. But the occasional implication of all forms of sensation must be kept in mind, and also the fact that in slight and slow or anomalous cases the sensory symptoms may long predominate, and for a time exist alone. The position of these symptoms (in the arms), and their frequent combination with spastic palsy in the legs, is a further characteristic—not, however, unequivocal.

Cervical pachymeningitis causes symptoms which, in general

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[*] Nissin, 'Arch. f. klin. Chir.,' Bd. xlv; 'Neur. Centralb.,' 1893, p. 100; Sokoloff, 'Deutsch. Ztschr. f. Chir.,' Bd. xxxiv; 'Neur. Centralb.,' 1893, p. 101; Galloway, 'Lancet,' 1891, vol. i.

[†] Contraction of the fields of vision, especially for colours, has been observed, but is not easy to explain (Dejerine and Turland).

[redacted] in the arms and the l syringomyelia ; but the affection anæsthesia involves all forms of less extent than the muscles only in their seat; pain is a the reaction of degeneration is Tumours of the spinal cord only especially of similar symmetry, and the diagnosis may then be But the resemblance is often generally arise from residual are frequently associated with the more rapid development of extension, that such a growth can of slighter chronic preceding described would justify the expecta- with the growth.

is sufficiently distinguished by the and in peripheral neuritis the reaction in the muscles should In "Morvan's disease," to be pre- may bear a close resemblance to those probable that they are due to a similar that it is to be recognised merely as a as a rule, however, the early loss of tactile prominence of trophic disturbance in the whitlows, enable the diagnosis to be affection of the cord that enters into is spinal hæmorrhage. It has been this condition that the hæmorrhage is cavity—as in the case shown in a cavity into which the blood has been the hæmorrhage has been preceded by syringomyelia, inquiry for which should extension occurs through a consider- cord, distinctly abolishing its central

much in seat and characters, as is glance at the illustrations given above; vary with the lesion. The diagnosis may in such cases, especially in the early when irritation and pain predominate, and ulated. It may be necessary to wait for the symptoms. Their progressive increase, always be taken into account; it fre- that suggests the nature of such a disease, the tendency of inflammatory affections

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to arrest and improvement. A peculiar difficulty is presented by the cases, not few, in which the first symptoms follow some influence—an illness, a blow, or even mental disturbance—that seems to be their cause, and suggests disease of very different nature. Often then, also, it is only by waiting and watching that a right opinion can be formed. When bulbar symptoms come on rapidly, the nature of the case can only be inferred from previous spinal symptoms.

The PROGNOSIS and TREATMENT of syringomyelia are subjects on which, unfortunately, its nature permits little to be said. If its existence can be recognised the malady is generally advanced, and its observed rate of progress is the only ground on which a forecast can be based. When the central functions of the cord are considerably impaired, and the symptoms are steadily increasing, it is rare for life to be prolonged for more than one or two years, and the subacute onset of considerable paralysis may herald a course that leads to death in a few months. On the other hand, a stationary condition may last for many years. Treatment can do nothing for the morbid state, nor is it likely that the progress of spinal surgery can afford the means of relief.[*] The severity of some of the individual symptoms may, however, be lessened by appropriate measures; especially trophic changes may be diminished by care and early treatment, bedsores may be prevented, cystitis guarded against, and if any acute paralysis occurs, the possibility of some recovery may suggest the maintenance of the irritability of the muscles by electricity, which cannot be expected to influence the slower atrophy. Pain may need the customary sedatives; but early pain, especially with the hyperæsthesia that depends on the intensification of the nerve-impulses at the irritated part, may be much lessened by the administration of cocaine, used as in tabes. Sudden pain in the spine may indicate commencing hæmorrhage, and should lead to the adoption of the posture and other measures appropriate to that grave affection.

ANALGIC PANARITIUM; MORVAN'S DISEASE.

By the term Morvan's Disease is generally known an affection that was called Panaritium Analgicum, or "Painless Whitlows," by the physician of Brittany, Morvan, who, in 1883, first described it.[†] Observations on it are still few,[‡] but, judging from two fatal cases,[§] it

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[*] In one case under my care the spinal canal was opened and the cavity drained, but without improvement.

[†] 'Gaz. Hebd.,' 1883, Nos. 35—44.

[‡] The chief writings are those of Louazel, 'La Maladie de Morvan, Paris, 1890; Joffroy and Achard, 'Arch. de Méd. exp.,' 1890; Charcot, 'Prog. Méd.,' 1890, Nos. 11 and 12; Häckel, 'Münch. med. Wochenschr.,' 1889.

[§] Gombault, quoted by Charcot; Joffroy and Achard, 'Arch. de Méd. exp.,' 1891.

Hence the state is sometimes discovered after death, when it has given rise to no symptoms, and its existence has not been suspected during life. In young children, indeed, it appears never to cause any definite symptoms, while the disturbance of function is variable in occurrence and degree—much more so than in character. The central position of the disease entails considerable uniformity in the distribution of its local consequences, and hence also in the derangement of function that results from simple increase in the lesion either by distension or growth. Hence the symptoms are often such that they have considerable diagnostic importance, although in a minority of cases they are less uniform in character, probably because the disease is unsymmetrical, or irregular in position or extension, whether the symptoms are due to the distension of a cavity, or to this combined with adjacent growth of tissue. It then often causes symptoms indistinguishable from those of a tumour of neoplastic nature. Moreover the precise level at which the chief morbid process takes place leads to a corresponding variation in the position, and therefore in the general aspect, of the symptoms it produces. The fact that the lesion is almost always greater at the cervical region causes the arms to be the most common seat of its manifestations, while the occasional invasion of the fourth ventricle by the associated growth, and the occurrence of coincident secondary changes in this part, explain the involvement of some of the cranial nerves that has been met with in a few instances.

The symptoms are usually of slow development, increasing gradually in the course of years. Their main features are twofold, of which one is clearly and certainly intelligible, while the explanation of the other is a matter of hypothesis, although also not difficult to understand, at least in part, if the opinions expressed in the chapter on the functions of the cord are correct. The two leading features are loss of sensibility, chiefly to pain and temperature, and muscular atrophy, resembling in characters that met with in the "progressive" spinal form. Of the two, the sensory loss is the earlier and more constant. The muscular atrophy is a necessary consequence of the damage to the anterior cornua from the compression they endure, when and where the central cavity or adjacent growth attains such a size as to damage them seriously, and they may be reduced to a mere zone between the cavity and the outer ring of white substance. The destruction of the motor nerve-cells involves the degeneration of the nerves and the wasting of the muscles. The loss of sensation is intelligible if the path of painful impressions is by the posterior commissure.[*] The enlargement of the central canal and disease in its vicinity can hardly fail to damage early and much the fibres that cross at the level of the chief lesion, and constitute the path from posterior roots not very far below. The course of thermal sensations the shoulder-joint has been observed.* The feet are occasionally, though rarely, the seat of anæsthetic ulceration; the legs have also been found weak, with excessive knee-jerk and foot-clonus (Häckel).

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[*] See p. 224 as to a tract in the grey commissure described by Ciaglinski ('Neur. Cent.,' 1896).

In the autopsy that was made by Gombault neuritis was found in both arms, and also a diffuse overgrowth of connective tissue in the posterior part of the grey matter of the cord and the posterior columns. This growth involved also the coats of the vessels, which were in places so thickened as to obliterate their cavity. Neuritis has also been invariably found in the extremities of the fingers when these have dropped off.

The features of the malady are so peculiar that, if they are known, the nature of a case cannot well be mistaken. Anæsthetic leprosy presents the closest resemblance, but in this the peculiar ulcerations are absent, and pigmentary alterations occur in the skin; while in Raynaud's disease there is not the peculiar loss of sensibility, nor an equal tendency to whitlows, and vaso-motor disturbance is a more conspicuous feature. In the peculiar malady known as "sclerodactyla" (a form of scleroderma that affects the fingers and face) there is no loss of sensibility or tendency to destruction of the finger ends. Simple syringomyelia is distinguished by the common absence of the whitlows, by the subordination, in time and degree, of the trophic changes to the other symptoms, and by the common preservation of tactile sensibility. But the distinction is probably not an absolute one. In Morvan's disease there is peripheral neuritis as well as syringomyelia, and to this is due its special features, and especially the early and intense trophic disturbance.†

SPINA BIFIDA.

Spina Bifida, or split spine, depends on a defect in the closure of the vertebral arches, which usually leads to the protrusion of the membranes as a sac, forming an external tumour, into which the lower part of the spinal cord often extends, normal or variously altered in conformation, sometimes even reduced to a neural lining of part of the wall. Rarely there is no external prominence—a form known as "spina bifida occulta."‡ The condition is most common in the lumbar region of the spine; sometimes is present at more than one place, very rarely in the whole length of the vertebral column. It is met with in about one child out of every thousand born (Chaussier). The disease is one of chiefly surgical interest, and therefore only an outline of the more important facts is here given. The spinal cord, whether normal or not, generally reaches lower than usual in the vertebral canal, as it does at the early period of development, when the morbid state is produced,—becoming adherent at its lower extremity and remaining so. In many cases the central canal of the cord is enlarged above, and the lower part of the cord may also be similarly distended.

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* Morvan, 'Gaz. Hebd.,' 1887, No. 34.

† The absence of any fundamental difference between this malady and syringomyelia has been pointed out by Bernhardt ('Verh. Vereins inn. Med.,' Berlin, January 19th, 1891) and Jolly (loc. cit.). But the additional element of neuritis must not be ignored.

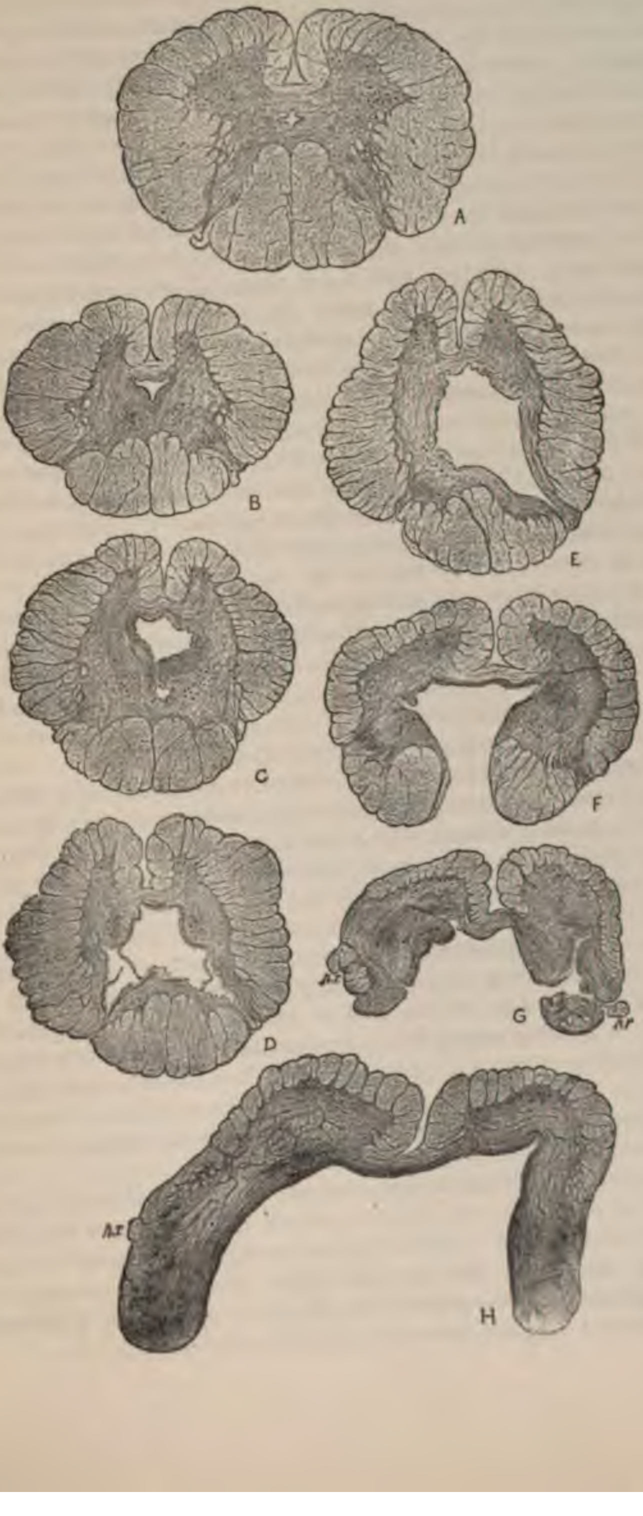
‡ The best accounts of spina bifida are in the "Report of the Committee of the Clinical Society" ('Transactions,' 1885), and Bland Sutton's "Lectures on Evolution in Pathology" ('Lancet,' February 25th, 1888).

The precise condition presents many variations, which can be, in part at least, understood by what is known of the process of development, a subject that has been very ably discussed by Bland Sutton. The essential element in spina bifida, the defect in the vertebral arches, depends on a deficiency in that ingrowth of the mesoblast from each side which should enclose the embryonal spinal cord, and form bone, after the superficial epiblastic layers have united to form the epidermis. A similar ingrowth of mesoblast forms the corium, muscle, &c. This apparently takes place in excess in spina bifida occulta, and leads to a curious condition, usually present—an excessive growth of hair over and about the affected part, which is generally the lumbo-sacral region. On the other hand, in many cases in which there is a tumour, this superficial mesoblastic ingrowth seems to be deficient, as well as that for the vertebral arches, so that at the upper part of the tumour there is an area in which there is no proper skin, but a peculiar shiny membrane destitute of hair. It is probably this superficial mesoblastic deficiency that permits the protrusion, and formation of an external tumour.

In spina bifida occulta the defect in the arches can be felt on palpation, either in the lumbar or sacral region, and the spinal cord extends lower than normal, as in the ordinary form. The latter, in which there is an external tumour, presents three chief varieties,

FIG. 190.—Spina bifida: sections of a spinal cord, from a case the symptoms and history of which I was unable to ascertain. In A, cervical region, the only abnormality is a large cruciform central canal and an unusually thick grey commissure. This continues through the dorsal region, at the lowest part of which there is a great change. In B, the canal is larger, and the necks of the posterior horns (and posterior vesicular columns) are united by much commissural tissue; in this, many vertical fibres could be seen, and others running from before backwards in the middle line. In C, an extension of the canal backwards has taken place, and it is bounded by a sinuous membrane; the outer part of the grey substance is atrophied. In D, the cavity has extended into each posterior horn, almost up to the surface of the cord, in part by a breaking down of tissue, because the membrane limiting the enlarged canal remains undestroyed. In E, still at the junction of the dorsal and lumbar regions, the cavity has receded from the left horn. In F, the division of the cord has taken place, not in the direction of the cavity in the horn (which is filled up), but in the middle line, at or close to the median septum. In G, a wider separation of the posterior columns has taken place, and a cavity has formed on the right side, which extends into the horn, and almost cuts off the posterior column. In H, the cord is spread out and formed part of the wall of the sac. The posterior nerve-roots (*p r*) mark the position at which the posterior horn comes to the surface. The grey matter is in many parts atrophied and translucent, and, in H, contains many large vessels. The septal lobulation on the surface of the cord is greater than normal.

FIG. 190.



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rem[illegible] of varieties.*

ab[illegible] forms, that without an external tumour has b
[illegible] met with, although it is probable that a knowledge
of [illegible] of the growth of hair in the lumbar region would le
an[illegible] of this state in many cases in which it is n
[illegible] Putting this form aside, simple meningocele a
[illegible] are both rare; the common form is mening
[illegible] which the cord, altered or intact, extends within the sa
[illegible]'s Committee found that this was the condition
[illegible] the cases in which there was an external tumour. Th
[illegible] the spinal cord is generally adherent to the posteri
[illegible] where its traction often causes a depression on th
[illegible] the membranous area, which, as already mentione
[illegible] be observed in the upper portion. The cord may agai
[illegible] extend downwards in the cavity, or it may b
[illegible] and lost in the wall, of which its tissue reall
[illegible] In this layer there is no distinction of grey an
In either case the lower nerves arise in the wall, an
[illegible] and then forwards, across the cavity, to their for
[illegible] The arachnoid always extends into the sac, and th
[illegible] contained within the subarachnoid space; sometime
[illegible] opening from which the fluid flows.

[illegible] central canal of the cord is not continuous with th
[illegible] be closed, even in the condition of syringomyelocele, i
[illegible] is dilated. In other cases the canal opens into th
[illegible] by only a small aperture, even when the lower part o
[illegible] into the wall, and the nerves course along the wal
[illegible] continuous with the arachnoid and superficial to th
[illegible] represents the cord-tissue from which they arise
[illegible] ordinary syringomyelia may exist in the upper part of th
[illegible] extension of the cord in the wall of the sac probabl
[illegible] developmental defect similar to that of the bony canal
[illegible] region the primitive canal has failed to close, so tha
[illegible] open posteriorly, and the two posterior columns may eve
[illegible] the cord may even be applied, in the form of a thic
[illegible] to the wall of the sac.

[illegible] although rare example of the involvement of th
[illegible] developmental effect is shown in Fig. 190, which illus
[illegible] the manner in which the latter may involve in some degre
[illegible] cord. In the cervical region the grey commissure is
[illegible] large, and the canal is cruciform, a shape which it present

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* Several other varieties are enumerated by Bland Sutton (loc. cit.).

at one period of development, and which is here persistent. This condition obtains throughout the dorsal region until, in the lower part, the tissue behind the commissure is so abundant as to unite the necks of the posterior horns. Below this the canal enlarges into a condition of syringomyelia, surrounded by the abnormal tissue which, as we have seen, is commonly associated with such enlargement; the canal is limited, moreover, by the sinuous membrane that is so often met with (see p. 633). The cavity extends also into the posterior horns, clearly by a process of disintegration, since in D the membrane lining the enlarged canal persists, and marks the limit of the canal and the extent of the simple disintegration. The progressive separation of the posterior columns is shown in F, G, and H. In the lowest part of the cord, the development of the posterior columns has been apparently hindered, as will be observed if G be compared with F. In the former there are also some fissures in the grey substance, formed apparently by disintegration. The grey substance of the cord is conspicuously wasted in the lumbar region, thin and translucent.

In rare cases there has been no proper development of the cord. It was represented by a mere nerve-like string in a child, who also had hydrocephalus, but lived five weeks. Another child of the same parents had a similar condition.*

SYMPTOMS.—If the cord is intact, symptoms may be absent. In the cases in which there is an external opening, and a constant escape of cerebro-spinal fluid, the child usually dies in a few weeks. In other cases the defective development of the lower part of the cord, or its distension in syringomyelocele, is manifested by an absence of function, amounting to complete paraplegia in severe cases, which also seldom live. Some cases, however, survive, with paralysis and wasting of the legs, and often absence of power over the sphincters. In slighter forms the paralysis and wasting have been partial, either existing from the first, or supervening at some later period, probably from the occurrence of damage to the nerve-roots or cord-tissue by increased tension, or by inflammation; of the latter abundant traces may be found after death, and various external influences may readily set it up. Some children, whose legs have appeared natural, have never been able to stand; possibly the defect in the posterior columns has given rise to a sort of congenital tabes. Even in the slighter forms, including spina bifida occulta, paralysis and atrophy have come on after some years, and even after adult life has been reached. Indeed, in the unsuspected forms without a tumour, there may be special danger of injury to the unprotected structures, and damage to the nerve-roots. The muscular paralysis and atrophy have been usually below the knees, and the muscles least prone to suffer are the tibiales antici. Hence talipes varus tends to occur. These muscles may alone persist, probably from the higher position of origin of their nerves (Remak). Another peculiarity of these cases is the tendency

* S. Wolfe, 'Phil. Rep.,' June 2nd, 1888.

to perforating ulcer in the feet, which has several times necessitated amputation. Chronic changes in the tarsal and metatarsal bones have also been met with, somewhat like those of tabetic arthropathy.[*] The ulcers suggest neuritis, which was found in one case; and, in this and others, the arteries have presented thickening of the muscular coat, even leading to their closure. These symptoms may be greater on, or confined to, one side, and on this the knee-jerk has been lessened.[†] These points of resemblance to tabes may also be due to the greater degree of exposure to damage of the posterior roots, as well as to neuritis, perhaps descending.

The treatment of spina bifida is purely surgical. That of the consequences just described is the same as for similar states in other diseases.

TRAUMATIC LESIONS OF THE SPINAL CORD.

Injuries to the cord fall within the province of the surgeon, but some account of them is needed because the subjects often afterwards come under medical observation, and because the cases present every gradation to the maladies that have been described in the preceding pages.

Causes.—In fractures and dislocations of the spine, the cord generally suffers compression or laceration, as already described in the chapter on injuries of the vertebral column. The cord may also be directly wounded by stabs and gunshot injuries, in which it may be divided completely or partially. Such injuries furnish a considerable proportion of the cases of unilateral lesion of the cord. In gunshot wounds the cord more often suffers from displaced fragments of bone than from the ball itself.

Far more common than direct injury is damage to the cord by concussion of the spine, either local or general. Gunshot wounds occasionally furnish examples of local concussion; a bullet may strike the spinal column and lodge in its vicinity, with the effect of causing immediate paraplegia, as complete as if the cord were divided, and yet it may be found that the spinal column has not been injured, and the cord is merely softened at the spot. Other causes of concussion are the fall of heavy bodies on the back, such as a beam of timber or a sack of corn, a blow on the back from some blunt weapon, a fall upon the back, either on a flat surface or on some projecting object. Less frequently the cord suffers from a general concussion of the body, in which the spinal column is not specially involved. The cervical region is occasionally damaged in falls on the head. Railway accidents are frequent causes of concussion of the spinal cord; the back may be struck with violence when the body is thrown from one side of the carriage to the other. Another common cause is a fall from a horse upon the back, or a fall downstairs, in which the spinal column is bumped against the edges of the steps. A violent bending of the spine may also injure the cord directly, especially in the more mobile cervical portion, where the region of the fourth and fifth vertebræ is especially apt thus to suffer (Thorburn).[*] The jar of the spine caused by a jump from a height may even be effective. Occasionally effects very similar to those which are produced by a blow on the spine are caused by a sudden contraction of the spinal muscles in some violent effort, a "rick of the back," as it is popularly termed. It is possible that, in such cases, the primary damage is sometimes to the vertebral ligaments and articulations, and that the cord suffers secondarily. The symptoms may only come on a few hours after the injury in such cases, as if effused blood compressed the cord, or the tension upon it and slight damage set up a graver inflammation. Thus a soldier, in a drunken quarrel, was extended by four comrades, two pulling on his feet and two on his arms, while a fifth sat on his back. No immediate effect followed, but the next morning there was complete paralysis of both arms and legs, which very slowly passed away.

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[*] In one case Sutton found the compact tissue very scanty, and the bones filled with semi-fluid fat; a deep ulcer reached a carious spot.

[†] Details of cases by Fischer, v. Recklinghausen, Brunna, and others, are quoted by Bland Sutton (loc. cit.). See also Delafosse, 'Thèse de Paris,' 1874; Remak, 'Berlin. kl. Wochenschrift,' 1885.

PATHOLOGY.—The anatomical lesions in cases in which the vertebral column is not injured vary much in different cases. Hæmorrhage is often found, sometimes outside the dura mater, sometimes on the inner surface of the membrane, in the pia mater, in the substance of the cord itself, very rarely into the central canal.[†] Occasionally the substance of the cord has been found lacerated when the vertebral column has not been injured. In many cases there is local softening, commonly yellow in tint, sometimes mingled with red, often involving the whole thickness of the cord, and occasionally extending, as central softening, through a considerable vertical extent. Under the microscope the usual products of inflammation are seen, sometimes with hæmatoidin crystals. Such softening may occur rapidly in severe local concussion, and be found complete a few weeks, or even a few days, after the injury.[‡] On the other hand, in some cases of complete paralysis, no lesion of the cord has been found, either with the naked eye or the microscope, a few days after the injury.* In other instances, in which the cord is examined some weeks or months after the accident, the signs of chronic myelitis are found, in scattered foci or more diffuse tracts, in the white columns or grey substance. The nerve-fibres are wasted, and the connective-tissue elements are increased in quantity, and in the early stages there may be a leucocytal infiltration about the vessels, dilatation of the capillaries, and minute extravasations, although none may have been visible to the naked eye. In the anterior cornua the motor nerve-cells may be damaged, sometimes swollen and vacuolated, or shrunken, and the anterior root-fibres may be degenerated. The grey matter is especially apt to suffer when the enlargements are injured; in the dorsal region the change may be confined to the white columns. Ill-defined cavities may exist in old cases, where the nerve-elements have perished. The usual ascending and descending degenerations may be found above and below the most damaged parts. Occasionally there are indications of meningitis, diffuse or disseminated, and sometimes confined to the dura mater.

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[*] 'Brain,' January, 1887.

[†] As in a case reported by Chucan and Wickham, 'Prog. Méd.,' 1887. Probably it is only when the canal is previously dilated that this occurs, as it may, without serious injury to the nerve-tissue.

[‡] Edmunds, 'Brain,' vol. vii, p. 103; Obersteiner, 'Wien. med. Jahrb.,' Bd. iii, 1879; Lochner, 'Bayer. Aerztlich. Int.-Bl.,' 1857, No. 42; Fromüller, 'Memorabilien,'

Symptoms.—The effects, immediate and remote, of injuries of the spinal cord extend over almost the whole range of symptoms of cord disease, and their variations in character and course are almost infinite. It is therefore neither practicable nor necessary to do more, in this place, than to describe their general characters. According to the difference in course, we may divide them into three classes. (1) Those in which the injury causes immediate and severe paralysis, due to instant damage to the spinal cord, the consequence of its laceration, compression, or concussion. (2) Those in which there are at first either no symptoms or only trifling disturbance of function, but in which grave symptoms come on a few days or weeks after the injury. (3) Those in which there are no early symptoms, or only slight and transient disturbance, but at the end of one or several months symptoms gradually come on, often such as indicate disease of some definite system or structure of the cord, degenerative in nature.

(1) The first class, in which the injury causes instant and considerable disturbance of function, includes the cases in which the cord is directly injured, and also some in which there is no visible sign of damage if the patient dies within a few days. In the former class there is often a manifest lesion of the spine, and the cases are, at least at first, purely surgical in their practical relations. In the latter the mechanical influence has apparently abolished the function of the nerve-elements. If such patients live longer, either quick recovery

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870, No. 12. In the last, described further on, softening was found thirty-two years after the injury.

* Fischer, 'Deut. Zeitschr. f. Chirurg.,' 1883, Bd. xix.

ensues, or local softening from disintegration of the fibres that are most damaged. It is an interesting fact that concussion may thus derange function. The absolute integrity of structure on early microscopical examination shows that the result is not due to any minute vascular lesion. The effect has been compared by Reynolds, not inaptly, to the demagnetisation of iron by a blow. Doubtless the influence is exerted on the molecular nutrition of the nerve-elements, and the possibility of recovery, or the subsequent structural disintegration, depends upon the degree of nutritional damage.[*]

The symptoms, in these cases of severe and immediate effect, are generally those of complete impairment of function. When the injury is direct and partial, such as a hemisection by an incised wound, or a partial bruise of the cord by a spiculum of bone being driven against it (as in the case mentioned at p. 236), the effect may be a partial (*e. g.* one-sided) derangement of function; but in most other cases there is abolition of all the conducting functions at the level of the injury. There is complete paraplegia, motor and sensory, with loss of power over the sphincters. The symptoms are thus those of a total transverse lesion at the affected level (see p. 269). When the cervical region is injured, the arms are necessarily affected according to its seat. Such cases often illustrate very clearly the relation of arm-function to the cord.[†] The fibres for the sympathetic are often implicated, especially those for the iris, causing inequality of the pupils. Reflex action below, abolished at first by shock, usually soon returns and becomes excessive, but remains absent if the lumbar enlargement is damaged, or is invaded by hæmorrhage; it may be again lost by the downward extension of secondary inflammation, which is equally effective when partial (in the posterior columns or grey matter) as when total, and is more difficult to detect in the former case.[‡] Loss of consciousness is occasionally produced by an injury which does not directly involve the head, as in the case mentioned on p. 656; vomiting is very common at the outset. The cases of direct injury often run a severe and rapid course; and when the secondary inflammation spreads downwards, or the lower part of the cord is injured (as

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[*] These conclusions have been recently formulated more definitely, but without essential extension, by Schmaus, partly from experiments on rabbits ('Münch. med. Wochensch.,' 1890).

[†] See especially Thorburn, 'Brain,' January, 1887.

[‡] This is the explanation of most cases of injury above the lumbar enlargement, with permanent loss of myotatic irritability, or of all reflex action, as in those described by Bowlby ('Lancet,' 1890, i, 1071). Such descending inflammation must not be confused with the descending degeneration in the pyramidal tracts which entails myotatic excess. The very rare exceptions mentioned on p. 264, in which such loss exists with no lesion to explain it, may be associated with the fact that myotatic irritability is sometimes lost in cases of cerebellar tumour. But the fact that superficial reflex action in the legs is never lost unless the lower part of the cord is diseased, is one of the most certain in pathology. Proof of such descending inflammation is given in the chapter on tumours (p. 617, note).

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[illegible] six months he could walk a
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[illegible] and when I saw him, six
[illegible] half a mile; the right leg
[illegible] was present in each leg.

[illegible] lowest part of the back re
[illegible] fall ends in the sitting
[illegible] nerves of the cauda equi

considerable a degree of recovery may be ultimately attained, it may be in the course of three or four years.

(2) In cases of the second class initial symptoms are absent or slight. After the accident the sufferer may not imagine himself injured, and may be able to walk some distance without inconvenience. In other instances there is tingling in the legs, or in all the limbs, immediately after the concussion, sometimes with some weakness of the limbs, more often with a feeling of stiffness. In the course of a few days graver symptoms come on, usually attended with spinal pain and tenderness, sometimes with some stiffness of the back, and often with pyrexia. Tingling in the limbs increases or develops, and is accompanied by weakness, which often goes on to complete paralysis in the course of one to four weeks. The symptoms vary in their character and distribution, according to the position of the morbid process and its extent. In some cases, in which the dorsal region is most affected, the symptoms are those of simple paraplegia, usually with spasm, and sometimes with early contraction of the muscles. In other cases, in which the grey matter of the enlargements suffers, there is scattered muscular atrophy in the limbs, often with indications of the degenerative reaction. Tremor in the limbs is conspicuous in some instances. A girdle-pain in the trunk, or sense of constriction in the limbs, is very common. The sphincters generally suffer, and the tendency to trophic changes is usually strong. The character and course of the symptoms is that of a subacute myelitis, and the meagre facts of morbid anatomy leave little doubt that this is the common lesion. Thus a lady was severely shaken in a railway collision. She seemed immediately after the accident to have suffered no injury, but in a few days paraplegia developed, and from its consequences she died six weeks after the accident. Throughout the dorsal region of the cord I found indications of subacute myelitis, chiefly in the white columns, varying in its extent in different regions, but most distinct in the pyramidal tracts.* Complete motor and sensory palsy, up to the level of the umbilicus, developed in the course of a week, after a fall downstairs in which the back struck successive steps. Yet the patient was able to walk immediately afterwards. The symptoms in these cases are sometimes unilateral. A man, driving under a low archway, leaned back to save his head, and his spine was pressed suddenly against the sharp edge of the seat-back. He felt but little immediate effect, but in the course of two or three days complete motor palsy of the left leg came on, accompanied by hyperæsthesia, but without any loss of sensation either in that leg or in the other. Power slowly returned at the end of three months, and was in time perfect. In some cases of this kind the symptoms are due

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* In the face of such a case as this, it is superfluous to discuss the question raised by some writers whether or not the cord can suffer concussion. Moreover it is certain that such an effect as was extreme in this case, may and must often occur in slighter degree.

not to direct damage to the cord, but to its compression by inflammatory products outside it, the result of injury to the vertebral column. A clergyman was thrown from his horse, and there was immediately sufficient weakness of the legs to prevent him from walking; this subsequently increased, so that at the end of a fortnight the right leg was completely paralysed, while the left retained considerable power. There was a girdle-pain at the level of the umbilicus, and a bedsore formed, but he slowly improved, and regained the power of standing at the end of eight months. Such symptoms may come on weeks after the concussion, especially when the effects of this are so slight as to be disregarded, and the patient leads his usual life.

When the damage involves the grey matter, the extent of the muscular wasting varies greatly. It seldom affects both arms and legs, but it is usually irregular in distribution, sometimes wide-spread, sometimes limited. Thus a young man fell from a horse and pitched on the head. He was stunned, and on recovering consciousness about two hours later felt "pins and needles" in both hands and pain in the back, followed by swelling of the neck and difficulty in moving it. The tingling ceased, but was followed by a sense of oppression about the shoulders, and persistent pain in the cervical region of the spine. When I saw him, two months later, there was some weakness of the right arm and wasting of the two outer interossei, and of that part of the long extensor which acts on the two outer fingers, with loss of faradic and preservation of voltaic irritability.

Sensory symptoms are prominent in some cases of this class. Besides the pain in the spine, to be presently mentioned, pains are often felt in the legs, various in character; still more frequent are spontaneous sensations of tingling, "pins and needles," and the feelings of defective sensibility described as "numbness." Various forms of hyperæsthesia are also common, with or without such subjective sensations. This may exist in any part, in the soles, feet, thighs, or not infrequently about the genitals. In the latter region, and about the anus, a sense of coldness is often complained of. In these regions there may be a distinct defect of sensibility, but this is slighter and less frequent than in the cases of the first class. Some degree of anæsthesia to touch may be associated with increased sensitiveness to stronger impressions. These sensory disorders may exist alone or be accompanied by motor weakness. Slight unsteadiness or definite inco-ordination is present in some cases, usually resembling that met with in ataxic paraplegia.

In cases which survive the acute stage there is usually slow improvement, which is often ultimately very great. In many cases recovery is incomplete, but improvement goes on for years, as it does in the cases of considerable damage to the cord already mentioned; the ultimate degree attained is commonly much greater than in cases of corresponding character and severity due to other causes than injury.

Some permanent symptoms are left, chiefly when the initial derangement of function has been severe, and has lasted for some time.

(3) In the cases of the third class, chronic symptoms slowly follow an injury at an interval usually of some months, and the cases have the aspect less of a traumatic lesion of the cord than of a primary cord disease, the relation of which to the injury is rather an inference from the sequence, coupled with the absence of other causes, than an obtrusive fact, as in the cases of the first and second classes. The symptoms are usually those of a definite system disease of the cord, less frequently those of a disseminated chronic myelitis of irregular distribution, still more rarely those of chronic focal myelitis. Primary spastic paraplegia, ataxic paraplegia, insular sclerosis, locomotor ataxy, and progressive muscular atrophy are the diseases which most frequently thus result. The fact that an injury is occasionally the cause of these maladies has been already mentioned in the account of their etiology, and the symptoms of such cases so far resemble those that are due to other causes that it is not necessary here to add anything to their history as already given.* We must assume that the shock to the nerve-elements causes a slow perversion of nutrition, which is only manifested by disturbance of function when it has gradually attained a certain degree. Occasionally other causes co-operate, capable of producing degeneration. Previous syphilis, or neurotic inheritance, can sometimes be traced, and it is a reasonable assumption that the predisposition thus arising may assist in rendering the traumatic influence efficient, or in augmenting its effects. Intermediate cases connect this class with the last, cases in which we must assume, from indications of chronic inflammation, that definite injury was produced at one or more spots. An instance of this, and also of focal myelitis, was presented by a man with permanent spastic paraplegia and an intense girdle-pain at the level of the umbilicus. These symptoms commenced three months after a jump from a table, and slight unsteadiness, with pains in the legs, connected the jump and the paralysis.

The several consequences of injury, which we have considered as occurring in separate form, are not only connected by intermediate cases, but are also sometimes distinctly combined. Thus an actual and immediate lesion of the cord may be associated with an early and severe increase in the symptoms from the development of subacute myelitis in other parts. The traumatic inflammation, at the spot damaged, often attains an intense degree, and runs a severe course, in consequence of the influence which may give rise to such inflammation, apart from a positive lesion; and, in both cases, the damage to the cord may be perpetuated by a degenerative tendency It is very common, for instance, for an injury to the cord, in those who have had

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* A case may, however, be mentioned in which typical tabes followed slowly a fall on the back, recorded by Barbour ('Journ. of Nervous and Mental Diseases,' March, 1891, Case IV).

not to direct [illegible] symptoms which subside, but not perfect[illegible]
matory products [illegible] of function may persist, and even incre[illegible]
column. A clergyman [illegible] in consequence of the influence of the previ[illegible]
immediately suffic[illegible] the nerve-elements prone to degenerate. Ag[illegible]
walking; this su[illegible] that the gouty diathesis is an unquestionable
night the right [illegible] often of the grave relapsing and spreading dis-
considerable pow[illegible] A concussion of the cord, which causes only slight
umbilicus, and a [illegible] the occurrence of this form of myelitis in
the power of [illegible] the symptoms of the later inflammation
may come on [illegible] after the injury (the effects of which have
of this are so slig[illegible] passed away), and are sometimes excited by some
usual life. [illegible] fresh concussion which would have had no

When the [illegible] individual. I have known the symptoms of insular
muscular wast[illegible] and spinal, to quickly follow traumatic paraplegia.
legs, but it is [illegible] remains to be considered, which is common to
sometimes limit[illegible] lesion of the cord, and is often severe and per-
on the head. [illegible] symptoms are slight—spinal pain. It may con-
two hours late[illegible] other symptoms have ceased, and probably
the back, follow[illegible] state of the nerves of the membranes or of
The tingling [illegible] column; often, probably, the pain depends on
the shoulders [illegible] joints and ligaments of the spine. It is essentially
When I saw [illegible] neuralgia. The pain may be felt at one or more
right arm and [illegible] it often extends through a considerable length of
the long ex[illegible] passes up to the occiput. It is occasionally
faradic and [illegible] sacrum, and may there have the character of a sense

Sensory [illegible] vague discomfort. The pain is associated with
Besides the [illegible] spine, usually deep-seated, chiefly developed at the
often felt [illegible] sometimes present also at other spots. It may
spontaneous [illegible] the features of a true neuralgia, may occur in
feelings of [illegible] induced by mental and other influences which do
forms of hy[illegible] the spine, as well as by exertion, posture, and other
tive sens[illegible] immediately influence the affected structures. This
not infr[illegible] called "spinal irritation."
the anu[illegible] traumatic lesions of the cord often acts also on the
there m[illegible] lesion may occur from the violence which affects
less fro[illegible] symptoms then co-exist with those of the spinal
anæsth[illegible] mask the latter during the early stage. More common,
strong[illegible] functional disturbance of the brain, the result, partly,
accomp[illegible] physical concussion, but chiefly of the mental shock
inco-ord[illegible] accident necessarily causes. The resulting condition
with i[illegible] the development and persistence of subjective sensory

In c[illegible] attention, maintained by concern, has a powerful in-
ment[illegible] on all forms of nerve-pain, and certainly aids in
is in[illegible] pain in the back, and even the tenderness which
cases[illegible] the spine. So marked is the influence of "nervous-
ultim[illegible] subjective symptoms, that it has been even maintained
corr[illegible] a large number of cases of concussion of the spine the

symptoms are of hysterical origin.* Well-marked symptoms of hysteria are sometimes manifested by these patients. But, on the other hand, it is necessary to avoid the danger of over-estimating the effect of mental influence, and of regarding, as entirely due to this, symptoms which are real, and are merely intensified by attention. The danger is especially great in cases of railway injuries, concerning which an unbiassed judgment is not easy to secure, and in which, when objective symptoms are absent, it is easy to minimise suffering, and attribute too much to the mental condition. The sinister influence of litigation on the intellect may be traced very widely.† I believe that it is rare for symptoms to be purely mental. It is often asserted by those employed for railway companies that subjective symptoms quickly subside when the sufferer's "claims" are settled, but it should be remembered that mental anxiety is a potent cause of diseases of the nervous system, and must be strongly opposed to recovery from genuine disorders. The occurrence of improvement when suspense is at an end is thus no proof in itself of the nature of the case, and its significance has been unquestionably over-estimated; moreover, in a great many individuals whom I have had an opportunity of observing long after they had received their "damages" (as the expression curiously runs) this subsidence had not occurred, and even the "sovereign balm" of substantial compensation has appeared to do very little for the relief of the sufferer.

These opinions have been formed from a study of cases other than those that involve litigation, in which no elements existed to bias the judgment, and from a comparison of these with many "railway" cases observed apart from forensic proceedings. Those who desire to learn what can be said on the subject of "railway spines," as they have come to be termed, when viewed from the opposite sides, will find abundant material for consideration in the writings of Erichsen ('On Concussion of the Spine,' London, 1875) and Clevenger ('Spinal Concussion, or Erichsen's Disease,' Philadelphia, 1889) on the one side, and of Page ('Injuries of the Spine and Spinal Cord,' London, 2nd ed., 1885) on the other. Scattered papers by J. J. Putnam, Walton, Spitzka, Buzzard, and others, more or less instructive, will be found epitomised in Clevenger's work, where indeed is collected the pith of almost all that has been written on the subject.

DIAGNOSIS.—The chief points in the diagnosis of traumatic lesions of the cord have been already incidentally considered. Immediate symptoms may be due to laceration, hæmorrhage, or to simple concussion, and the diagnosis between these is not always possible at first. If there are immediate symptoms of a partial lesion, these

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* J. J. Putnam, 'Boston Med. and Surgical Journal,' 1883, Sept. 6th.

† Not many years ago it was customary for the "experts" who gave evidence on behalf of railway companies to deny that the spinal cord could be injured if the legs were unwasted. Although the opinion dare not now be expressed, its significance is not without analogies at the present day.

already indicated. It is important t
any symptoms beyond the simpl
or "creeping" sensations may be of
persistent sensation of "pins and
A definite sense of constriction
organic disease, and so is a well-
the muscles on the two sides. The
the excess is slight, general, and
significance if the diminution is
groups of muscles, such as the flexor
Any impairment of power o
of great diagnostic importance; loss of
is of little value, since this fun
mental anxiety and preoccupation. A
is most significant when it is partial.
is strong presumptive evidence of
of the knee-jerk is of little value; al
indicates some changes in the nutrition
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features but of non-traumatic origin. If the symptoms are slight or moderate in degree approximate recovery may be anticipated, although slight symptoms often endure for a very long time. Indeed, in many cases recovery, although approximate, is not perfect. The patient is never quite as strong, never becomes quite as capable of exertion, as before the injury. A cautious prognosis should also be given whenever there is the late and gradual onset or increase of symptoms that suggests a degenerative process. Such degeneration presents far less tendency to arrest or subsidence than do the earlier lesions. As a rule the sooner symptoms occur the better is the prospect of ultimate improvement or recovery, provided they are not so severe as to be incompatible with life.

Treatment.—The early treatment of these cases, and much of the later treatment of those in which the spinal column is injured, is purely surgical. The points of medical character alone need special mention, and many of these have been anticipated in the preceding pages. In all cases in which spinal symptoms are present immediately after an injury, however slight those symptoms may be, absolute rest should be insisted on for some days or weeks, according to the severity of the early symptoms. This is necessary on account of the secondary inflammation, which, as we have seen, so often occurs. The treatment of developed symptoms must be conducted on the same general principles as in cases of myelitis; the details need not be here repeated. If there is muscular wasting, it is important that the nutrition of the muscles should be maintained by electrical stimulation, since a very considerable amount of ultimate recovery may be anticipated, and it is important to keep the muscular tissue as far as possible in a condition to respond to the nerve-power when this returns. If there is reason to believe that there is inflammation of the membranes, or inflammatory effusion outside the cord, compressing it, mercury may be given, but this condition is probably much more rare than might be anticipated. The influence of mercury on inflammation of the substance of the cord is doubtful. The chief element in treatment is patiently to permit time to do its work, and the tissues to slowly regain such integrity of structure and function as is possible; meanwhile preserving the patient from all influences likely to interfere with the process or to set up other mischief, such as cystitis or bedsores, which would entail fresh danger. There is, however, one therapeutic measure that is of unquestionable value in the treatment of the later stages, especially of the cases in which the recurring symptoms suggest a relapsing myelitis as the sequel of injury; and that is the repeated application of a mild "actual cautery" on each side of the spine opposite the affected region. Several applications should be made, with or without an anæsthetic. It is not desirable to lessen the pain by cocaine, because this is likely to interfere with the influence of the proceeding, the beneficial character

will show that, in a case of purely hysterical paraplegia, the morbid functional condition is cerebral; the brain-centres which act on the legs are at fault, but the condition of the functions of the cord itself may be absolutely normal. The spinal motor centres are in a state of inactivity because the related cerebral centres are inactive, but this is no more a diseased condition of the cord than is its corresponding functional state during physiological rest. Hysterical paraplegia will be described, with other palsies of like origin, in the chapter on hysteria—a malady for which the term "functional" is often employed in various ways, as a convenient euphemism.

At the same time, it must be remembered that there is no sharp line of demarcation between functional derangement and nutritional changes. As already mentioned, no functional state can exist without leaving behind it some corresponding change in the finer molecular nutrition of the structures; and if functional derangement of lower structures results from that of higher cerebral centres, and is maintained for long, the change in nutrition that results may be considerable, and may be so definite as to persist even after its cause has ceased to act. Moreover the general enfeeblement of defective nutrition of the nerve-elements is a potent cause of this functional disturbance, and at once facilitates and augments such effects. The process may be observed in many cases not only of hysteria, but also of other derangements of the cerebral functions, and it is seen also in lower centres as an effect of pain.

Isolated disturbance of functional centres in the cord does, however, sometimes occur. It is seen in the conditions of inhibition which are designated "reflex paralysis." Such palsies were once thought to be common and persistent, but it has been proved that many, and it is probable that all, cases of considerable and prolonged palsy, formerly thought to be of reflex origin, are due to organic disease, either primary in the cord or secondary to an ascending inflammation of nerves. But transient paralysis sometimes occurs, which cannot be otherwise explained than as an inhibition of a spinal centre, due to peripheral irritation. Such, for instance, is the curious inability to pass urine which sometimes follows an operation on the anus,—the division of a fistula, for instance, or the removal of hæmorrhoids. The inability may continue absolute for several days. Transient weakness of one arm is said sometimes to follow an operation for empyema (Lépine), but the fact that the weakness may be attended by choreoid movements in both arm and leg of one side (Weil) makes it probable that the influence is exerted on a cerebral rather than on a spinal centre.* Considerable paraplegia was thought to be sometimes a reflex effect of disease of the bladder, or of a calculus or other organic disease in the kidney, but it is probable that such cases are always of organic nature. Ascending neuritis, reaching the cord,

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* Paralysis and wasting of serrati magni and infra-spinati muscles have, however, followed double empyema (Cayley, 'Clin. Soc. Trans.,' 1898, vol. xxxi).

secondary to morbid states of the cerebral motor centres, or form part of diseases of wider range in connection with which they are described.

Other cases which may be regarded as functional are those in which symptoms, commonly subjective in character, result from some morbid blood-state. The conditions which most frequently have this effect are gout and diabetes. Occasionally there is definite failure of power, lasting for a few days or weeks, without objective symptoms, and passing away. But the most common symptoms from this cause are sensory and subjective,—feelings of tingling and formication in the legs, dull aching, and sometimes actual pain; this is usually transient, but occasionally continues for some days or weeks, various in position, but in gouty cases often felt in the heels. Such symptoms due to morbid blood-states occur chiefly during the second half of life. Those of gouty origin, like other symptoms of the same class, are especially common in persons who inherit a tendency to gout, but have not suffered from attacks of definite arthritis.

In the diagnosis of functional disorders the first element is the exclusion of any sign of positive disease, and the second is the discovery of some morbid state capable of giving rise to the functional derangement, or of associated symptoms unequivocally functional in character. The first of these is that of primary and paramount importance, which, alike in practice and reasoning, should precede the others. The various points involved in the diagnosis of hysterical affections are described in the chapter on that disease in Vol. II; one only need be mentioned here—the importance which attaches to the state of myotatic irritability in the legs, and the great difficulty in ascertaining it presented by many of these cases. Permanent excess shows that there is more than functional derangement; although it does not exclude the latter, it indicates changes in nutrition, and will be returned to presently. The cases in which the knee-jerk is apparently absent present a special difficulty. It is never really absent in functional disorder (except when the centres are exhausted by violent convulsion), any more than it is in health. Failure to obtain it is due to the inability of the patient to relax the muscles, which may render attempt after attempt futile, until, in some fortunate moment, relaxation is secured and a characteristic jerk is obtained. The expedients that facilitate the attempt have been already mentioned (p. 21), and the subject has been sufficiently discussed in the account of the diagnosis of tabes.

The treatment of functional disorders is that of the morbid states to which they are due, or of which they form part, and does not need special description. That of the symptoms due to toxic influences is necessarily the removal of the condition which causes them, but they may be to some extent relieved by sedatives, of which bromide of potassium, cocaine, Indian hemp, cimicifuga, and small (7 or 10 gr.) doses of chloralamide are the most effective.

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tions in spinal nutrition in that disease are apparently the result of the impressions on the peripheral nerves. In women such a condition is often associated with uterine or ovarian pain, and with sacral pain apparently of uterine origin. It is possible that the condition we have been considering is sometimes secondary to such uterine pain, which acts in a manner somewhat analogous to that in which joint inflammation acts, and that this is the explanation of many of the cases in which a reflex disturbance of the functions of the cord has been supposed to be of uterine origin. The condition is also often associated with spinal pain and tenderness, which may have a similar influence.

In the diagnosis of these nutritional affections of the spinal cord it is important to remember that they may be closely simulated by analogous changes in the peripheral nerves, constituting the slighter degrees and forms of polyneuritis. The chief points in the distinction have been described in the account of that disease. It is in the parenchymatous forms, and especially in the sensory varieties, that the danger of confusion is apt to arise. Symmetry in the distribution of the subjective sensations, or positive anæsthesia, and their localisation in the extremities, are the most important features of neuritic change.

Connected with this distinction is another, of not less importance. Nutritional changes, equally with the simpler functional derangement, may be the result and expression of the action of some toxic blood-state, and all that has been said in connection with mere alteration in function applies also to the class now under consideration. Toxic influences cannot, indeed, act for long without leading to changes in nutrition, which may speedily attain a degree that amounts to visible structural disease. A search for such an agency should never be neglected in any case in which the symptoms have not followed an adequate cause, and do not form part of a general state of the same nature. But it must be remembered that the peripheral nerves are, as a rule, more susceptible to toxic influences than is the spinal cord, and this fact gives additional weight to any indications that it is from these that the symptoms proceed. Lastly, symptoms suggestive of mere changes in the finer nutrition of the nerve-elements are frequently the earliest indications of organic disease of one or another of the types already described, and in all recent and progressive cases their general character and aspect must be carefully considered, and compared with the known features of the various organic diseases.

The cases with such slight but persistent symptoms as have been just described vary much in their features, and may present all sorts of slight symptoms, in the utmost diversity of degree and combination. To consider them in detail would be to describe an indefinite series of individual cases, no two of which are identical. The precise degree and manner in which the nerve-elements suffer may, it is evident, be almost infinitely various. Their corresponding manifestations present features, combinations, and gradations that entirely baffle an attempt to designate them, or even to perceive definite types about which they can be grouped. These are some of the cases of disease which exemplify the failure of types in practical work, the error involved in the attempt to give names, and the paramount importance, in the process of diagnosis, of considering what morbid condition the symptoms indicate, and of dealing with each case as a new problem, *sui generis*, whether or not it is ultimately found to be one of a familiar series. This method, moreover, conducts the practitioner at once to the morbid processes that need treatment, and should determine the character of his efforts to alter that which is at fault.

It is unfortunately not common for the indications of nutritional disturbance of the cord, when they have been long established, to pass away altogether, although some degree of improvement can often be secured; and in many cases, especially in those of brief duration, approximate restoration to a normal state may take place.

The treatment necessarily varies according to the precise condition that exists, and the causes on which it depends. The removal of these as far as possible, and of any influence likely to depress the nervous system, must be the first consideration. It should be remembered that no part of the nervous system is unaffected by mental depression, and that impairment of the general health may render impossible recovery from local disease. From these general principles the details of the treatment of individual cases, too various to be here described, may readily be deduced. Therapeutic measures that are thus reasoned out and determined on by the indications of the special case are far more likely to be successful than those that are simply taken from a description of treatment. The only points that need special mention are, first, the importance of securing mental tranquillity by producing the conviction, when possible, that no grave disease exists or is impending, and with it the disregard of those slighter sensations of discomfort that become more aggressive and disabling the more they are noticed. At the same time it is important that the patient should avoid all over-exertion, all risk of falls or chills, and should be especially careful in regard to sexual intercourse, which often has a peculiar effect in augmenting the symptoms; and in the unmarried, perfect continence should be enjoined. Often, indeed, in the case of men, the mental preoccupation causes an apparent failure of sexual desire, which itself is a source of concern—generally groundless. Reassurance on this point frequently helps to secure the desired mental state. Equally important is it to disabuse the sufferer's mind of the idea that seminal loss is weakening—an error widely prevalent; the truth being that any depressing influence comes only from the associated nervous action. But it is important to lessen undue frequency of nocturnal emissions, and for this nothing is so effective as Milton's remedy, large doses of the tinct. ferri perchlor. (♏xx or xxx) three times a day, coupled with the avoidance of lying on the back.

The relief of disabling pain, by the measures described in the chapter on neuralgia, and by the treatment of any local morbid state, is indispensable. Rest from work, the improvement of the general health, and the administration of nervine tonics, such as arsenic, phosphorus, quinine, strychnia, and the like, are the chief other points in treatment. A course of massage, or of gentle faradisation of the muscles, is also often useful.

APPENDIX.*

THE MUSCLE-SPINDLE.

In 1862 Kölliker first described the muscle-spindle, and in the following year Kühne added considerably to the description given by Kölliker. Both these investigators considered that the muscle-spindle was a stage in the development of muscle and nerve, and although in the following years a considerable number of investigators on the subject published their researches, it was not till 1888 that Kerschner argued the sensory nature of the muscle-spindle. In 1893 Ruffini described an annulo-spiral nerve termination within the spindle, but it remained for Sherrington in 1894 to prove by experiment that the nerve supplying the spindle passed up in the posterior root and definitely to settle the sensory nature of the muscle-spindle.

Distribution.—Muscle-spindles have been found in nearly all skeletal muscles of the body, excepting the eye muscles and the diaphragm. They are abundant in the small muscles of the hand and in the arm muscles, and are more numerous in the belly of the muscle than near the tendon. In relation to this statement it is well to recognise that the musculo-tendon organs are found to be numerous in the region of the tendon. The muscle-spindle is present at all ages from the fourth month of fœtal life onwards.

Size.—The size of the muscle-spindle varies considerably, not only in the same muscle but also with regard to the length of the muscle, and the age of the subject, being longer in the adult than in the child,

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* The following account of the muscle-spindle has been kindly written by Dr. F. E. Batten, whose able and industrious researches on the subject render this expression of his views most valuable.

fine fibres, some of them measuring only ·008 mm. At a certain point in the equatorial region some of the muscle-fibres lose their transverse striation and nuclei appear in the substance of the muscle-fibre. These nuclei gradually increase in number till they completely fill the fibre, then after a short distance they become less numerous, and the muscle-fibre again resumes its striation. At the equatorial region the muscle-fibres do not fill the whole spindle, but lie to one side. As they pass to the distal end of the spindle they become joined again and pass out of the spindle as two or three fibres. It will be seen from the above description that a transverse section of the spindle may at a given point in the equatorial region seem to contain no

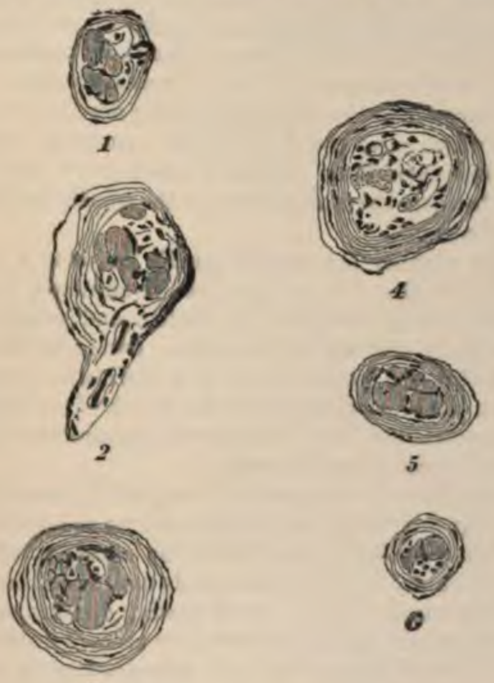

FIG. 192.—Normal spindle of child in transverse section. Magnified 250 diameters.

1. Section near the pole. The areas marked with vertical lines are muscle-fibres in transverse section.

2. Nerve entering into spindle. Many of the dark bodies in contact with the muscle-fibres are nerve-fibres.

3. Nearer the equatorial region the sheath has acquired considerable thickness.

4. At the equatorial region the muscle-fibres have undergone considerable modification, and are no longer recognisable as such.

5. The spindle near the opposite pole; the muscle-fibres have resumed their natural appearance.

6. At the distal pole of the spindle.

regard to their nerve-supply and the character of their muscle-fibres.

In *peripheral neuritis* certain changes have been described within the muscle-spindle both as regards the muscle-fibres and also as regards the nerves; in other cases, however, they have been shown to be perfectly normal.

In *tabes dorsalis*, although the external form of the spindle and the nerves passing to it have been found to be normal, certain changes have been described in the intra-fusal muscle-fibres. The evidence, however, on this point is insufficient at the present time to justify us in regarding the changes described as constant in this disease.

References.—Sherrington, 'Journal of Physiology,' vol. xvii, p. 237, 1895; Ruffini, 'Journal of Physiology,' vol. xxiii, p. 190, 1898; Batten, 'Brain,' vol. xx, p. 138, 1897.

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